Clinical Protocols in
PEDIATRICS

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Second Edition

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Our Families

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Preface

We come across various clinical scenarios in our day-to-day practice that requires careful analysis and on the spot decision making. Each of these cases presents with various possibilities. In order to properly manage these cases one must have logical approach. This book primarily deals with management of such situations. This is neither a textbook nor a book dealing with cases in intensive care or critical care. The effort starts at trying to understand the severity and gravity of each individual case, resuscitate at emergency department, order some investigations to come to a working diagnosis, then manage appropriately according to the diagnosis and refer to the appropriate specialty either as out-patient or admitting to ward or intensive care unit (ICU) after stabilization.

Clinical Protocols in Pediatrics is approach-based, algorithmic and naturally ready to use. Even with sound knowledge, working in a busy emergency may be stressful and tiresome. Going through a textbook or descriptive text may be time consuming and mind boggling too. We need "at a glance" format which can be accessed by emergency physicians, emergency fellows, trainees, pediatric postgraduates and even practicing pediatricians.

The book deals with ideas, which can be practised in any reasonably good hospital set-up. It also covers emergency situations that can be dealt with both by emergency physicians and pediatricians. Chapters on Dermatology, Orthopedics and even Psychiatry discuss topics where there is a clear boundary what as a pediatrician and emergency specialist should do and when he should refer to other specialists. A lucid approach has been made for relatively new pediatric trainee or emergency trainee.

It was an absolute pleasure for us in writing this book. We are grateful to all the contributing authors for their valuable inputs. We are indebted to our alma mater the Kanchi Kamakoti Childs Trust Hospital, Chennai, Tamil Nadu, India, where during our postgraduation, we used to encounter innumerable cases during our long hours in the emergency room. Perhaps, it was the driving force for this venture.

A supportive family is the backbone of any work. We are fortunate to have understanding parents, wives and children. We are grateful to our parents Ashish and Jaya Choudhury, Arun and Anjali Bandyopadhyay, spouse Sudarsana Choudhury and Munmun Bandyopadhyay, children Sayondeep Choudhury and Chandrika Bandyopadhyay, for their constant help and encouragement.

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All attempts have been made to acknowledge the sources of information. Inadvertent omission if any is regretted.

We are open to suggestions and criticisms. Feel free to mail us at drjaydeep_choudhury@yahoo.co.in and sendjayanta@yahoo.com.

Jaydeep Choudhury
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1

General Emergency

Jayanta Bandyopadhyay, Jaydeep Choudhury

1.1 APPROACH TO A CHILD IN PEDIATRIC EMERGENCY DEPARTMENT

Jayanta Bandyopadhyay, Jaydeep Choudhary

Children presenting to the emergency department are more difficult to assess than adults. Smaller children are more difficult to assess as most of the time they present with nonspecific signs and symptoms. A strong clinical judgement is the cornerstone for determining the severity of the condition in children.

Warning Symptoms in History

1. Child with persistent lethargy, who are not playful and not smiled over a few hours
2. Child with prolonged irritability or inconsolable child
3. Child taking less than 50 percent of normal fluids
4. No urine output for 6 hours or more
5. Seizure, cyanosis, pallor or apnea
6. Parental concern out of proportion to child's illness.

Warning Signs in Examination

1. Respiratory distress, grunting
2. Tired or looks sicker than usual illness
3. High pitched cry
4. Drowsy
5. Floppy
6. Pale

7. Alteration in vital signs
8. Low saturation
9. Signs of shock
10. Full or bulging fontanelle
11. Non-blanching petechiae or purpura
12. Bilious vomiting.

Decision-Making in Pediatric Emergency

It is the whole process of balance of history, examination, knowing when to trust the parents, intuition and maintaining objectivity. Sometimes the features are glaring and decision making is very easy, at other times there is no definite pointer but somehow one feels that everything is not right and these are the difficult situations. But in pediatric emergency probably a bit of over doing is better than taking a chance. When in doubt, it is better to admit a patient for observation.

Normal Vital Signs

The vital signs like respiratory rate and heart rate are crucial points in examination. Unlike adults the children are unique in this sense as both this parameters change with age. The normal weight, respiratory and heart rate for children of different age groups and the differentiating features between sick and well children are shown in Tables 1.1.1 and 1.1.2, respectively.

TABLE 1.1.1

Normal weight, respiratory and heart rate in children of different age groups

Age	*Weight (kg)*	*RR / min*	*HR/ min*
Birth	2.5–3.5	40–60	100–160
3 months	6	30–50	100–160
6 months	8	30–50	100–160
1 year	10	30–40	100–160
2 years	12	20–30	100–150
4 years	15	20	80–130
6 years	20	16	70–120
8 years	25	16	70–110
10 years	30	16	60–100
12 years	40	16	60–100
14 years	50	16	60–100

TABLE 1.1.2

Differences between sick and well children

	Well child	*Sick child*
General behavior	Plays with toys	Not playful
	Interacts with parents	Not much interaction
Mood	Normal	Irritable
	Alert, smiles	Unhappy
	Consolable if cries	Cries more than usual, inconsolable
Wakefulness	Awake	Sleepy
	Normal sleep pattern	Difficult to waken
Tone	Normal	Floppy
Vital signs	Afebrile	Fever
	RR and HR normal	Tachypnea
		Tachycardia

Things to be done in Emergency Room

1. Get the relevant history from the parents or the reliable informers only.
2. Do not allow a crowd to gather—it jeopardizes the whole process and management.
3. All the children attending the emergency department should be attended as an emergency situation—at least immediately by the nurses if the doctor is busy with some other patient.
4. Competent nurses are as important in an emergency department as the doctors.
5. Most of the time, the seriousness of the condition can be assessed immediately by a gross inspection.
6. None of the children should be dismissed without proper assessment.
7. If there is any doubt after clinical assessment, it is always better to admit a child brought to the emergency department. If beds are not available, the child should be stabilized first before referring to other hospital.
8. Emergency equipment should be available, such as oxygen supply, sucker apparatus, bas and mask ventilation.

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1.2 EARLY ASSESSMENT AND EMERGENCY TRIAGE

Jayanta Bandyopadhyay

Triage includes early patient assessment, priority rating, first aid, control of patient flow, assignment of correct area of care, initiation of diagnostic measure and liaising with relatives and health care professionals. Many early hospital deaths can be prevented by an accurate triaging and thus instituting quick treatment. Following is an example of triage categories though the process may be never perfect leaving the chance of patients having more serious illness than the category suggests.

Priority 1—Severe Life-threatening Emergency

Management should start immediately and needs to be shifted to resuscitation area equipped with resuscitation backup.

1. Cardiac or respiratory arrest
2. Hypovolemic shock
3. Severe trauma (pediatric trauma score 8 or below)
4. Coma (GCS 10 or below)
5. Status epilepticus
6. Anaphylaxis with cardiorespiratory decompensation.

Priority 2—Acute Medical Problems or Distress

Management should start within 10 minutes and the child should be shifted to acute care area with arrangement for basic life support.

1. Severe respiratory distress
2. Febrile child with signs of sepsis
3. Developing petechial rash
4. Venomous bite
5. Fractured shaft of femur
6. Fractured cervical spine with cord damage
7. Multiple fractures
8. Burn more than 10 percent.

Priority 3—Semiurgent Acute Medical Problems

Should be attended in 10 to 30 minutes. Also should be kept in acute care area.

1. Moderate respiratory distress
2. Potentially venomous bite in asymptomatic patient
3. Febrile infant younger than 3 months

4. Febrile child with irritability, drowsiness or pallor
5. Moderate dehydration
6. Limb fractures.

Priority 4—Nonurgent Acute Medical Problems

Should be attended in 30 to 60 minutes.

1. Recent seizure activity
2. Mild dehydration
3. Suspected limb fractures with no deformity and minimal pain
4. Nontraumatic limp.

Priority 5—Condition is Stable and will not Deteriorate as a Result of Waiting

1. Uncomplicated laceration
2. Febrile older child in absence of signs of sepsis
3. URTI in absence of fever.

Though this is an outline, the list can not be exhaustive and each patient needs individual assessment. A continuous assessment and monitoring of already categorized patient is also necessary by triage station from patient deteriorating into more urgent category while waiting for treatment. Remember, watchful trained eyes looking for basic signs of life like respiratory rate, heart rate, work of breathing, sensorium, pulse volume, capillary refill, cyanosis and saturation combined with clear idea about differential diagnoses and potential outcome of early institution of treatment are the essence of successful triage program.

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2

Resuscitation

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2.1 CARDIOPULMONARY RESUSCITATION

Jayanta Bandyopadhyay, Joshi Anand Karketta, Jaydeep Choudhury

A sudden cardiac arrest is uncommon in children. Cardiac arrest is usually the terminal event of progressive respiratory failure or shock. Any emergency set up should be well equipped with procedures for cardiopulmonary resuscitation (CPR).

Airway

1. Mild neck extension is preferable (Child's head and occiput are proportionately large causing neck flexion). One can use a folded towel placed under the neck and shoulder.
2. Open the airway by head tilt—chin lift method. If cervical injury is suspected, open the airway using a jaw thrust without head tilt.
3. Clear airway from secretions, vomits and remove foreign bodies. In neonates and infants this can be effectively done with bulb mucus sucker (Fig. 2.1.1).
4. Oropharyngeal and nasopharyngeal airways for maintaining an open airway.
    i. Oropharyngeal airway in unconscious patient, i.e. with no gag reflex. The size is determined by the distance from the central incisors to the angle of the mandible.
    ii. Nasopharyngeal airway is better tolerated than oral airway by patients who are not deeply unconscious. The size is determined by the distance from the tip of the nose to the tragus of the ear.

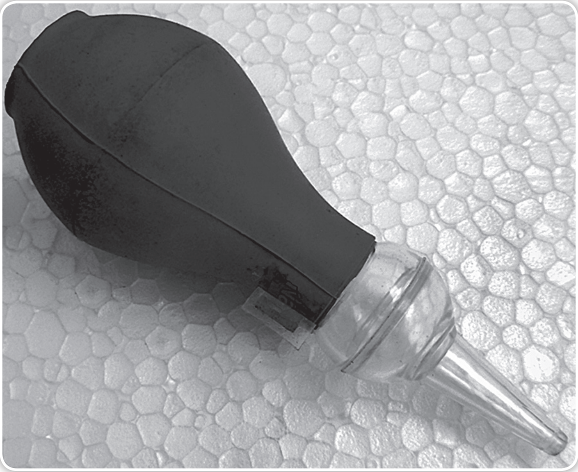

Figure 2.1.1 Bulb mucus sucker

Breathing

1. Use 100 percent oxygen during resuscitation.
2. Bag-mask ventilation can be as effective as endotracheal intubation:
    - i. Use a self-inflating bag with a volume of 450 to 500 mL (Fig. 2.1.2)
    - ii. Maintain oxygen flow of 15 L/min into a reservoir attached to a bag
    - iii. The mask should fit over the mouth and nose to provide a tight seal and avoid any air leakage.
3. Ventilation through an endotracheal tube (ETT) (Fig. 2.1.3).
   Size for children 1 to 10 years of age is determined as ETT internal diameter (mm) = (age in years/4) + 4.
4. Laryngeal Mask Airway (LMA): When endotracheal intubation is not possible LMA is an acceptable adjunct for experienced providers.

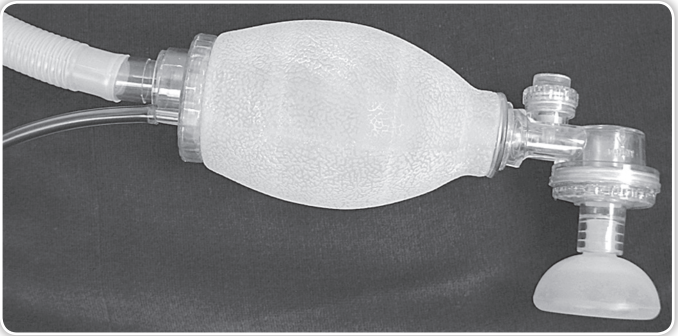

Figure 2.1.2 Bag and mask ventilation

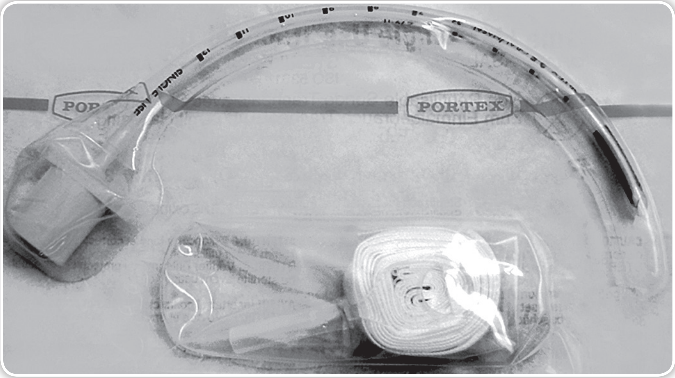

Figure 2.1.3 Endotracheal tube

To Minimize Gastric Inflation

i. Avoid excessive peak inspiratory pressures (e.g. ventilate slowly and watch chest rise, deliver only the volume needed to produce visible chest rise)
ii. Apply cricoid pressure to obstruct the esophagus
iii. Pass NG tube after intubation because a gastric tube interferes with the gastroesophageal sphincter, allowing possible regurgitation.

Excessive Ventilation is Detrimental Because:

i. It impedes venous return and, therefore, decreases cardiac output, cerebral blood flow and coronary perfusion by increasing intrathoracic pressure
ii. Causes air trapping and barotraumas in patients with small airway obstruction
iii. Increases the risk of regurgitation and aspiration.

Circulation

1. Check pulse (brachial artery in infants—carotid or femoral artery in children)
2. Start cardiac compressions when heart rate <60 beats/min with signs of poor perfusion. Two thumb—encircling hands chest compression and bag and mask ventilation is shown in Figure 2.1.4.

Characteristics of Good Chest Compressions

1. "Push fast" at a rate of 100 compressions/min
2. "Push hard" to depress the chest 1/3 to 1/2 of the anterior-posterior diameter of the chest
3. Release completely to allow the chest to fully recoil after each compression
4. Minimize interruptions in compressions.

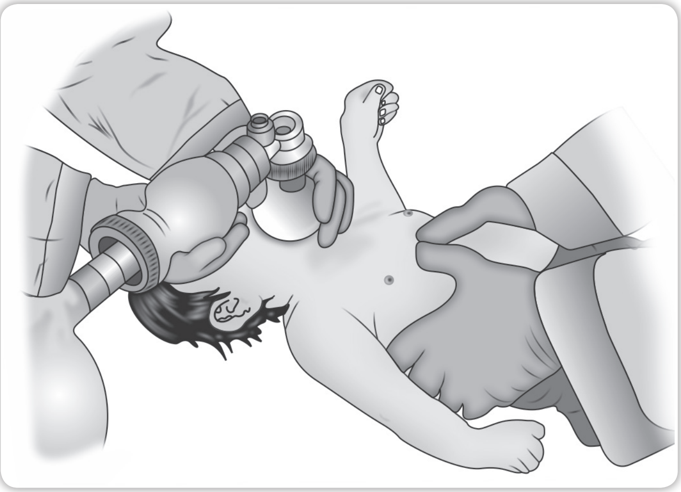

Figure 2.1.4 Two thumb—encircling hands chest compression and bag-mask ventilation

Recommended Chest Compression: Ventilation Ratio

1. One rescuer: Cycles of 30 compressions and two breaths
2. Two rescuers: Cycles of 15 compressions and two breaths
3. When an advanced airway is established (e.g. ETT or LMA):
    i. Give continuous chest compressions without pauses for breaths
    ii. Give 8 to 10 breaths/minute
    iii. Check rhythm every two minutes and change the compressor role to prevent fatigue and deterioration in quality and rate of chest compressions
    iv. The switch should be done in less than five seconds to minimize interruption in chest compression
    v. If the pulses are present but no breathing, give 12 to 20 breaths per minute (1 breath every 3 to 5 seconds).
    The method of chest compression is shown in Table 2.1.1.

Vascular Access

If one cannot achieve a reliable access quickly, an intraosseous (IO) access should be established.

Fluids

1. Isotonic crystalloid solution to treat shock (20 mL/kg of normal saline as quickly as possible). Repeated boluses may be necessary.

TABLE 2.1.1

Method of chest compression in infants and children

Cardiac compressions	*Infants*	*Children*
Rate/min	100	100
Depth	1/3 of AP chest diameter	1/3 to 1/2 of AP chest diameter
Site	Lower half of the sternum not over the xiphoid (below intermammary line)	Lower half of the sternum not over the xiphoid
Technique	2 fingers technique. OR 2 thumb—encircling hands technique (preferred)	Heel of one hand or two hands technique

2. Hypotension—Systolic blood pressure less than 5th percentile of normal for age:
    i. <60 mm Hg in term neonates
    ii. <70 mm Hg in infants
    iii. <70 mm Hg + (2 × age in years) in children 1 to 10 years
    iv. <90 mm Hg in children >10 years of age.
3. Glucose containing fluids are not indicated during CPR unless hypoglycemia is present.

Drugs

1. Drugs are preferably administered through IV or IO route than by ETT.
2. If vascular access cannot be established, the following drugs can be given via ETT – "LEAN" – lidocaine, epinephrine, atropine and naloxone. Flush with a minimum of 5 mL normal saline followed by 5 assisted manual ventilations. If CPR in progress, stop chest compressions briefly during administration of medications.
3. Epinephrine dose in cardiac arrest = 0.01 mg/kg of 1:1000 epinephrine as the first and subsequent IV doses.

Medications for pediatric resuscitation and arrhythmias that should be available in pediatric emergency and medications to maintain cardiac output and for postresuscitation stabilization are shown in Tables 2.1.2 and 2.1.3, respectively.

TABLE 2.1.2

Medications for pediatric resuscitation and arrhythmias

Medication	*Dose*	*Remarks*
Adenosine	0.1 mg/kg (max 6 mg)	Monitor ECG
	Repeat dose 0.2 mg/kg (max 12 mg)	Rapid IV/IO bolus
Amiodarone	In pulseless VT or VF—5 mg/kg IV/IO	Monitor ECG and BP
	Repeat up to 15 mg/kg, maximum 300 mμg.	Use with caution when administering with other drugs that prolong QT
	Infusion—25 μg/kg/min for 4 hours followed by 5–15 μg/kg/min continuous.	
Atropine	0.02 mg/kg IV/IO 0.03 mg/kg ET* repeat once if needed minimum dose 0.1 mg maximum single dose in child 0.5 mg, in adolescent 1 mg	Higher doses may be used with organophosphate poisoning
Calcium chloride (10%)	20 mg/kg IV/IO (0.2 mL/kg)	Slowly
Epinephrine	0.01 mg/kg (0.1 mL/kg 1:10 000) IV/IO 0.1 mg/kg	May repeat every 3–5 min
	(0.1 mL/kg 1:1000) ET* maximum dose 1 mg IV/IO; 10 mg ET*	
Glucose	0.5–1 g/kg IV/IO	10% dextrose diluted with 5–10 mL/kg 25% dextrose diluted with 2–4 mL/kg
		50% dextrose diluted with 1–2 mL/kg
Lidocaine	Bolus 1 mg/kg IV/IO	
	Maximum dose 100 mg	
	Infusion 20–50 μg/kg/min ET* 2–3 mg	
Magnesium sulfate	25–40 mg/kg IV/IO over 30 minutes, may be given faster in Torsades pointes	
	Maximum dose 2 g	
Naloxone	<5 years or ≤20 kg 0.1 mg/kg IV/IO/ET*	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1-15 μg/kg)
	≥5 years or >20 kg 2 mg IV/IO/ET*	
Procainamide	15 mg/kg IV/IO over 30–60 minutes adult dose 20 mg/min IV infusion up to total maximum dose 17 mg/kg	Monitor ECG and BP use caution when administering with other drugs that prolong QT
Sodium bicarbonate	1 mEq/kg/dose IV/IO slowly	After adequate ventilation

* flush with 5 mL of normal saline and follow-up with 5 ventilations

TABLE 2.1.3

Medications to maintain cardiac output and for postresuscitation stabilization

Medication	*Dose*	*Comment*
Dobutamine	2–20 μg/kg/min IV/IO‡	Inotrope, vasodilator
Dopamine	2–20 μg/kg/min IV/IO‡	Inotrope, chronotrope; renal and splanchnic vasodilator in low doses; pressor in high dose
Epinephrine	0.1–1 μg/kg/min IV/IO*	Inotrope, chronotrope, vasodilator in low doses; pressor in higher doses
Norepinephrine	0.1–2 μg/kg/min*	Inotrope, vasopressor
Sodium nitroprusside	1–8 μg/kg/min‡	Vasodilator; prepare only in D5W

‡6 × body weight (in kg) = mg of drug to add to 100 mL D5W then, an IV rate of 1 mL/h delivers 1 μg/kg/min of drug.
*0.6 × body weight (in kg) = mg of drug to add to 100 mL D5W, then, an IV rate of 1 mL/h delivers 0.1 μg/kg/min of drug.

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2.2 COMA

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Clinical status of a patient can be divided into four types—alert, lethargy, stuporous and coma. Coma is defined as the state of unarousable unresponsiveness.

Neurological Examination

Level of Consciousness

Modified Glasgow coma scale (GCS) as shown in Table 2.2.1 has been used in children and infants to assess the duration and depth of altered consciousness.

Attitude and Posturing

Resting position and spontaneous movements should be noted. Hemiparesis, focal motor seizure, or any asymmetrical finding indicates structural brain lesion, whereas symmetrical lesion is usually seen in metabolic coma. Decorticate rigidity indicates structural lesion of cerebral hemisphere or

TABLE 2.2.1
Modified GCS

Response	*Score*
Eye opening	
1. Spontaneous	4
2. To speech	3
3. To pain	2
4. None	1
Best verbal response	
1. Smiles, oriented to sound, follows object, interacts	5
2. Consolable cry	4
3. Inconsistently consolable cry	3
4. Inconsolable cry, restless, agitated, unaware of environment or parents	2
5. No response	1
Best motor response	
1. Spontaneous movement/obeys verbal command	6
2. Localizes to supraocular pain	5
3. Withdraws	4
4. Abnormal flexion to pain	3
5. Abnormal extension to pain	2
6. None	1
Grading—13–14 Mild, 9–12 Moderate, <8 severe	

diencephalons whereas decerebrate rigidity indicates upper brainstem lesions. Medullary lesions cause flaccidity.

Respiration

Abnormal respiratory pattern in coma helps in localization:

i. Cheyne-Stokes respiration—Bilateral hemispheric or diencephalic lesions.
ii. Apneustic respiration—Upper pontine lesions.
iii. Cluster breathing—Lower pontine or upper medullary lesions.
iv. Ataxic respiration—Lower medullary lesions.
v. Central neurogenic hyperventilation—Midbrain or upper pontine lesion.
vi. Kussmaul breathing—Metabolic acidosis.

Brainstem Function

i. *Pupillary response:* Assuming the visual pathways to the lateral geniculate body are intact, assessment of the pupillary responses is important in localizing the site of coma and separating structural from toxic/metabolic causes, as pupillary responses in the latter are intact.
    a. Unilateral fixed dilated pupil—Uncal herniation or rupture of posterior communicating artery aneurysm
    b. Unilateral constriction—Horner's syndrome
    c. Bilateral pinpoint pupils—Pontine lesion or opiate poisoning
    d. Bilateral midposition—Nonreactive in midbrain lesion and Reactive in metabolic causes
    e. Bilateral dilated and fixed—Severe midbrain damage.

ii. *Ocular motility:* It is an important clinical indicator of the site of lesion in coma.

    If there is spontaneous eye movement, look for the following.

    a. Conjugate spontaneous roving movements indicate an intact brain stem
    b. Conjugate lateral deviation—Look towards the lesion indicates cerebral hemispheric lesion and away from the lesion is seen in brainstem damage
    c. Ocular bobbing indicates bilateral pontine or cerebellar lesion
    d. Skew deviation occurs with posterior fossa lesions
    e. Persistent adduction in paresis of VI cranial nerve
    f. Persistent abduction in paresis of III cranial nerve
    g. Dysconjugate gaze in horizontal plane indicates intoxication
    h. Downward deviation of the eyes indicates bilateral thalamic or subthalamic lesions and metabolic coma.

        If there is no spontaneous eye movement, then ocular motility can be tested by oculocephalic reflex (Doll's eye maneuvre) and oculovestibular reflex (Caloric testing). The methods of the tests are shown in Table 2.2.2.

TABLE 2.2.2

Methods of testing oculocephalic and oculovestibular reflexes

Oculocephalic reflex	*Oculovestibular reflex*
Eyes move conjugately to the opposite direction to head movement: Normal	Nystagmous with fast component away from site of stimulation: Normal
Lateral gaze palsy: Brainstem lesion	No nystagmus or dysconjugate response:
No movement: Pontine lesion	Brainstem lesion
Full deviation towards the stimulated side: Cerebral hemispheric lesion with intact brainstem	

iii. *Fundoscopic examination:* Assess for papilledema and any hemorrhage.
iv. *Other cranial nerve reflexes:* Corneal reflexes (V and VII cranial nerves); gag and cough reflexes (IX and X cranial nerves) should also be assessed. Corneal reflex is retained until coma is very deep.

Signs of meningeal irritation: Presence of meningeal signs indicates meningitis or subarachnoid hemorrhage and parenchymal bleed with intraventricular extension.

Clinical differentiation of toxic, metabolic or infectious causes of coma from structural causes of coma like supratentorial and infratentorial coma are shown in Table 2.2.3.

Investigations

1. Blood sugar: In all patients presenting with coma or altered sensorium, blood sugar or blood glucose should be tested with Dextrostix. Treatment should not be delayed of symptomatic hypoglycemia awaiting this result
2. Full blood count, malarial parasite in peripheral smear
3. Blood culture
4. Consider coagulation screen before considering a lumbar puncture
5. If hypoglycemic: Measure blood ketones. Insulin, growth hormone and cortisol may be measured if possible.

TABLE 2.2.3

Differences between metabolic and structural coma

Toxic/metabolic/infectious	*Supratentorial lesions*	*Infratentorial lesions*
1. Confusion precedes motor signs	1. Initial focal signs	1. Preceding brainstem dysfunction
2. Pupillary reactions preserved	2. Rostral to caudal progression	2. Sudden onset
3. Symmetrical motor responses		3. Cranial nerve palsies
4. Asterixis, myoclonus		4. Early respiratory disturbances
5. Hyper/hypoventilation		

6. Urea and electrolytes
7. Blood gas, including arterial lactate
8. Liver function tests: Consider serum ammonia in case of suspected hepatic coma, Reye's syndrome or urea cycle defect
9. Thyroid function tests
10. Urinalysis: Consider toxicology screen of urine and blood if indicated
11. CT scan or MRI brain: If trauma or clinical suspicion of child abuse, focal neurodeficit. Initial normal CT does not rule out an evolving lesion of an infection or metabolic disorder
12. CSF analysis: Lumbar puncture (LP) is contraindicated in presence of coma (GCS <9), raised intracranial pressure or unstable clinical state. If meningitis is suspected but LP is contraindicated, start antibiotics
13. Cervical spine imaging: Protect neck until injury has been excluded by standard criteria in cases of trauma or possible trauma. It is often not possible to exclude cervical spine injury in a comatose child. May need to CT scan of upper cervical spine in trauma
14. MRI provides better visualization of brainstem and cerebellar structures, venous sinuses, diffuse disorders, for example in inborn errors of metabolism or hypoxic encephalopathy
15. EEG: Serial EEG is more useful. Relatively specific in herpes simplex encephalitis. It helps in detection of nonconvulsive status epilepticus as a cause of coma.

Management

Early appropriate supportive care is essential to avoid preventable secondary insults and optimize the neurological outcome. Evaluation and stabilization of the patient's airway, breathing and circulation (ABCs) must proceed simultaneously with assessments of the depth of coma and the presence of raised intracranial pressure (ICP). Any rapidly correctable cause of coma must be immediately corrected.

Airway

Ensure adequate airway. Use airway adjunct oropharyngeal airway, nasopharyngeal airway, LMA or intubate if unable to maintain airway. If GCS <8 and clinical circumstances do not suggest improvement or imminent herniation, then intubation is indicated to secure airway. Even if spontaneously breathing with normal gas exchange, many comatose children will benefit from intubation, especially if they have intracranial hypertension.

Breathing

Give oxygen, monitor oxygen saturation, and assess rate and pattern of breathing. Supplemental oxygen is indicated for hypoxia, but should not be given routinely to comatose children with normal circulation and oxygen saturation.

Circulation

Assess for signs of shock and treat as indicated. The aim of fluid therapy in raised ICP is to maintain adequate cerebral perfusion pressure (CPP).

CPP = Mean arterial pressure – Intracranial pressure.

Give normal saline or Ringer's lactate to maintain BP and perfusion. What must be restricted are hypotonic fluids such as 1/5th normal saline in 5 percent dextrose (Isolyte P). The dextrose will be metabolized with a resultant hypotonic fluid that can exacerbate cerebral edema and ICP. Enteral feeds should be started at the earliest.

Hypoglycemia

Bolus of 10 percent dextrose 5 mL/kg IV, followed by a 10 percent dextrose infusion at 4 mL/kg/h (7 mg/kg/min) with close monitoring of blood glucose.

Hyperglycemia

Due to stress or diabetic ketoacidosis.

Drugs

If opioid toxicity is suspected—Naloxone 0.1–0.8 mg/kg IV (maximum dose 2 mg). Avoid flumazenil, which may induce convulsions in mixed overdoses, particularly if tricyclic antidepressants have been taken. Isolated benzodiazepine overdose does not cause significant respiratory depression and children are best managed with simple observation.

Specific Therapy

After stabilization, a rapid approach to diagnosis is imperative so that specific therapy can be given.

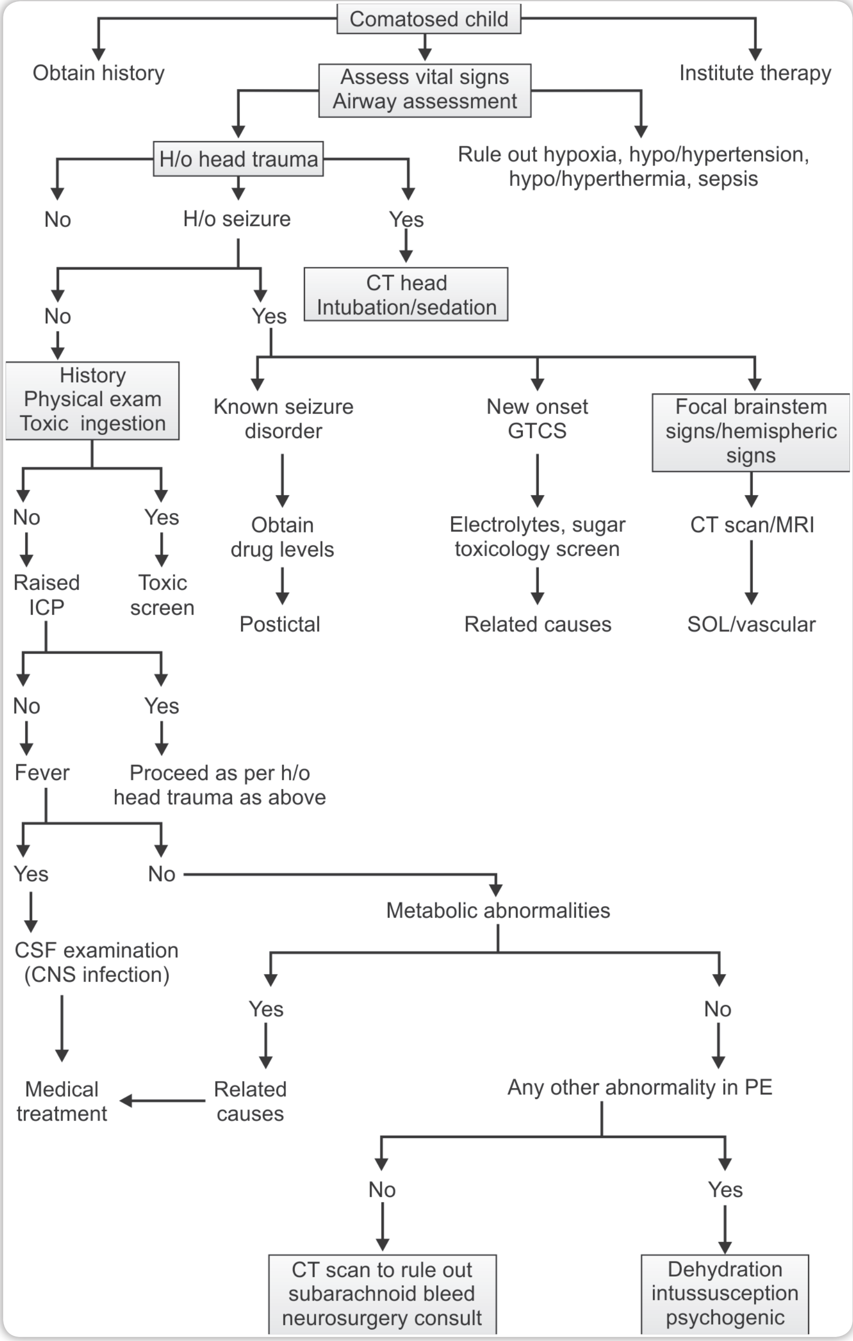
1. Known poisoning—specific antidotes
2. Hypertensive encephalopathy—antihypertensives
3. Space occupying lesions—neurosurgery.

The stepwise approach to diagnosis and management of coma in children is shown in Flow chart 2.2.1.

Emergency Management of Raised Intracranial Pressure

GCS <9 and Cushing's triad of hypertension, bradycardia, abnormal respiration and/or fixed, dilated pupil(s).

Flow chart 2.2.1 Algorithm for the diagnosis and management of coma in children



1. Airway: Oxygenate and intubate.
2. Ventilation: Avoid hypercapnea with an aim to maintain a pCO_2 of 30 to 35 mm Hg
3. Osmotic therapy: Use 3 percent NaCl, 3 mL/kg IV given as a rapid infusion or use mannitol 0.5 g to 1.0 g/kg (2.5 to 5 mL/kg of 20% mannitol). Mannitol is indicated acutely for patients in whom there is a strong clinical suspicion of raised ICP or imminent herniation. In the hypotensive/hypoperfused patient, hypertonic saline may be the osmotherapy of choice for reducing ICP while maintaining MAP/CPP
4. Sedation and pain relief: For example, morphine and midazolam
5. Elevate head to 30°
6. Control fever—Give paracetamol intragastrically or rectally 10 to 15 mg/kg q 4 hourly
7. In raised ICP refractory to medical measures, surgical decompression has been shown to improve survival and functional outcome.

Antiseizure Medications in Coma

Convulsions can cause massive increases in CBF, consequent increase in ICP, can lead to secondary brain damage and may precipitate or be precipitated by cerebral herniation. Apart from generalized tonic clonic seizures, some comatose children may have nonconvulsive seizures manifesting with subtle signs such as eyelid twitching, eye deviation or nystagmus. If in doubt, empiric treatment of seizures may be justified and can result in improvement of consciousness.

Choice of Empiric Antimicrobials

If a CNS infection is suspected in a febrile child presenting in acute coma and seizures, empiric antimicrobial should include acyclovir in addition to a third generation cephalosporin until further confirmatory tests are available. The need for empiric antimalarials should be carefully assessed.

Prognosis

Recovery from coma depends primarily on the cause rather than depth. Metabolic coma carries the best prognosis, while prolonged stupor following global hypoxic event has a poor prognosis. Early rehabilitation in most cases is often very much rewarding.

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2.3 ANAPHYLAXIS

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Anaphylaxis is a rapidly evolving multisystem allergic reaction which is potentially life-threatening and may progress to shock, lower and upper airway obstruction or all of them. Clinical features may be as mild as simple urticaria to upper airway obstruction, hypotension and death.

Management

Airway, Breathing and Circulation (A, B and C)

In case of anaphylaxis, maintenance of patent airway and oxygenation is of utmost importance. The child should lie flat on a firm surface with raised foot end to facilitate blood supply to vital organs.

Treatment of Choice

Injection Aqueous Epinephrine 1:1000 dilution, 0.01 mL/kg deep intramuscular. The maximum dose should not exceed 0.3 to 0.5 mL. The same dose may be repeated every 15 minutes. Though, subcutaneous route may also be employed but skin perfusion is often poor due to peripheral vasoconstriction and hence deep intramuscular route is preferred. The same may be given via endotracheal tube.

In case of serious reaction an intravenous infusion of epinephrine may be started at 0.1 mg/kg/min to sustain a systolic blood pressure of 80 mm Hg.

Hypotension

Rapid intravenous infusion of normal saline, sometimes colloids may be required. Dopamine may sometimes be used for maintaining the blood pressure. Infusion of epinephrine and norepinephrine may be used to counter widespread vasodilatation and capillary leak.

Antihistaminics

Diphenhydramine 1 mg/kg intravenous, intramuscular or per oral may be given and repeated 4 hourly along with ranitidine 3–6 mg/kg/day in 2 or 3 divided doses.

Systemic Corticosteroid

Its role is debatable and questionable. It may be of help in the late phase of allergic reaction. The following drugs may be used:

Methylprednisolone 2 mg/kg intravenous followed by 1 mg/kg every 6 hourly.

Hydrocortisone 10 mg/kg intravenous followed by 5 mg/kg every 6 hourly.

Salbutamol

Nebulized salbutamol 0.15 mg/kg with normal saline may be repeated every 20 min to counter the bronchospasm.

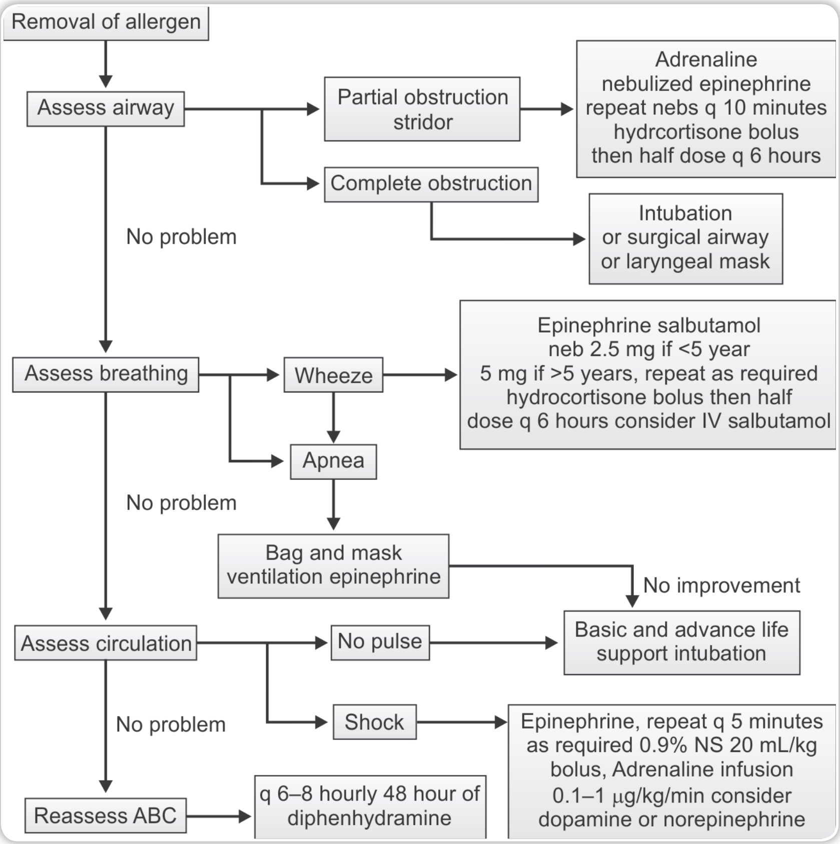
The doses of various drugs in anaphylaxis are shown in Table 2.3.1 and the Flow chart 2.3.1 shows the step-wise management of anaphylaxis.

TABLE 2.3.1

Pharmacotherapy of anaphylaxis

Medication	*Dose*
Epinephrine (1: 1000)	10 µ g/kg IM and 0.1–1 µg/kg/min IV as infusion
Norepinephrine	0.1–1 µg/kg/min
Hydrocortisone	4 mg/kg
Dopamine	2–20 µg/kg/min IV
Diphenhydramine	5 mg/kg/day PO in 3–4 divided doses

Flow chart 2.3.1 Algorithm for anaphylaxis treatment


Removal of allergen
Assess airway
Partial obstruction stridor
Adrenaline nebulized epinephrine repeat nebs q 10 minutes hydrcortisone bolus then half dose q 6 hours
Complete obstruction
Intubation or surgical airway or laryngeal mask
No problem
Assess breathing
Wheeze
Epinephrine salbutamol neb 2.5 mg if <5 year 5 mg if >5 years, repeat as required hydrocortisone bolus then half dose q 6 hours consider IV salbutamol
Apnea
No problem
Bag and mask ventilation epinephrine
No improvement
Assess circulation
No pulse
Basic and advance life support intubation
No problem
Shock
Epinephrine, repeat q 5 minutes as required 0.9% NS 20 mL/kg bolus, Adrenaline infusion 0.1–1 µg/kg/min consider dopamine or norepinephrine
Reassess ABC
q 6–8 hourly 48 hour of diphenhydramine

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2.4 FLUID AND ELECTROLYTE BALANCE

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Fluid and electrolyte balance is a very important aspect of pediatric emergency management. A large number of children present to the emergency department with dehydration due to various etiology. The commonest cause of dehydration in children is acute gastroenteritis.

General Guidelines for Rehydration

1. Better under hydrate than over hydrate—Do not exceed 100 mL/kg for deficit replacement.
2. Care for the kidney function is crucial; it will take care for minor miscalculations. This is done by providing 20 mL NS/kg in case of hypovolemia.
3. An overweight infant is misleading:
    i. Signs of dehydration are late and can be missed
    ii. They may be already in shock when recognized
    iii. Hypernatremia is a risk.
4. An underweight infant:
    i. Has poor tolerance to the usual calculations
    ii. Needs calories more than water and sodium
    iii. Signs of dehydration are exaggerated.
5. A drowsy child with gastroenteritis is critical:
    i. Associated CNS infection
    ii. Brain edema (hyponatremia)
    iii. Brain dehydration (hypernatremia).
6. Abdominal distension with gastroenteritis is always abnormal:
    i. Perforated appendix
    ii. Intussusception
    iii. Septicemia, late NEC
    iv. Hypokalemia.

Rehydration in Acute Gastroenteritis

While managing a dehydrated child, appropriate fluid therapy is vital. Various intravenous fluids are available. It is important to use that intravenous fluid which is closest to physiological milieu. Thus, one should be familiar with the compositions of commonly used rehydration solutions as shown in Table 2.4.1.

The type of rehydrating solution is chosen according to its sodium content. Carbonated drinks and apple juice should not be used for rehydration as they do not contain sodium and have 10 to 12 percent sugar concentration.

TABLE 2.4.1

Composition of commonly used rehydration solutions and plasma

	Content	*Osmolality*
Normal saline (NS)	Na = 150 mmol/L	300 mOsmol/kg
	Cl = 150 mmol/L	
0.45 NS 5% D	Na = 75 mmol/L	428 mOsmol/kg
	Cl = 75 mmol/L	
	Glucose = 50 g/L	
0.22% NS	Na = 37 mmol/L	
	Cl = 37 mmol/L	
8.4% $NaHCO_3$	HCO_3 = 1 mmol/mL	
	Na = 1 mmol/mL	
Human plasma	Na = 145 mmol/L	290 mOsmol/kg
	K = 4.5 mmol/L	
WHO-ORS	Na = 90 mmol/L	320 mOsmol/kg
	K = 20 mmol/L	
	Citrate = 30 mmol/L	
	Glucose = 110 mmol/L	

Principles

1. Correction of hypovolemia or shock
2. Replacement of deficit
3. Provision of maintenance
4. Allowance for ongoing losses (if any)
5. Attention to the sodium status.

Hypovolemia or Shock

20 mL/kg NS over 30 to 60 minutes can be repeated if there is no response. For adolescents 10 mL/kg.

Deficit Replacement

Volume replacement depends on degree of dehydration as shown in Table 2.4.2. Sodium loss in various states of dehydration is shown in Table 2.4.3.

Apart from hypernatremia the sodium content of a rehydrating solution for deficit replacement is 80 to 100 mmol/L

TABLE 2.4.2
Volume replacement in dehydration

	Younger children	*Older children*
Mild	50 mL/kg	30 mL/kg
Moderate	75 mL/kg	50 – 60 mL/kg
Severe	100 mL/kg	70 – 90 mL/kg

TABLE 2.4.3
Sodium loss in various states of dehydration

Status	*Approximate sodium loss*
Isonatremia (acute)	100 mmol/L
Isonatremia (chronic)	70–80 mmol/L
Hyponatremia	120 mmol/L
Hypernatremia	40–60 mmol/L

Maintenance Requirements

Following deficit replacement, fluid balance is continued by maintenance therapy.

Volume

Water or calorie requirement is shown in Table 2.4.4.

Sodium

Requirement is 2 to 4 mmol/kg/day

Potassium

Requirement is 2 to 3 mmol/kg/day.

Ongoing Loss

If IV hydration is considered, you can successfully rehydrate most children with the following solutions.

TABLE 2.4.4
Water or calorie requirement

Weight	*Water (or calorie) requirements*
Birth–10 kg	100/kg
11–20 kg	1000 + 50 /kg above 10
21–30 kg	1500 + 20 /kg above 20

1. Normal saline (150 mmol Na/L)
2. 0.45 NS in D5 percent (75 mmol Na/L) and add 4 percent KCl (0.5 mmol/mL).

Start with 20 mL NS in any child with more than moderate dehydration, discount them from the deficit. Then, as per following calculation.

1. 6 to 8 hours: Deficit + 1/3 maintenance.
2. 6 hours: Deficit + 2/3 maintenance.

It has to be remembered that Na content in deficit fluid is 80 to 120 mmol/L and that in maintenance fluid is 30 mmol/L. The following solution is satisfactory.

0.45 NS = 75 mmol/L sodium.

To each liter add 20 to 30 mmol K = 40–60 mL 4% KCl. Which comes to each 500 mL fluid add 10–15 mmoL K = 20–30 mL 4% KCl.

Example

A 12 months old child with acute gastroenteritis is admitted with severe dehydration. His weight is 10 kg.

Total fluid needed is as follows:

Deficit = 10 × 100 = 1000 mL

Maintenance = 10 × 100 = 1000 mL

Step 1: IV 0.9% NS 200 mL over 60 minutes.

Step 2: IV 0.45 NS in 5% D 500 mL + 4% KCl 25 mL. Volume—700 mL over 7 hours.

This provides the remaining deficit (400) + 1/3 of the maintenance (300) = 700 mL.

Step 3: IV 0.45 NS in 5% D 500 mL + 4% KCl 25 mL.

Rate 60 mL/h over 16 hours. This provides 1000 mL (approx.), which covers the remaining deficit + 2/3 of the maintenance.

Hyponatremic Dehydration

By definition serum sodium less than 130 mEq/L is hyponatremia. Low sodium concentration in serum may be due to water retention, sodium retention or both. Consequently, the extracellular fluid volume may be low, normal or high.

Serum sodium less than 125 mEq/L is associated with lethargy, nausea and vomiting. When serum sodium is less than 115 mEq/L neurological manifestations like seizures and coma develop.

Asymptomatic Hyponatremia

It is better to explain with an example. A 12 months old child with acute gastroenteritis is admitted with severe dehydration. His weight is 10 kg.

Step 1: IV 0.9% NS 200 mL over 60 minutes (same as in isonatremia).

Step 2: IV 0.45 NS in 5% D 500 mL + 25 mL 4% KCl (Rate/duration as in isonatremia).

Step 3: IV 0.45 NS in 5% D 500 mL + 4% KCl 25 mL (Rate/duration as in isonatremia).

Symptomatic Hyponatremia

1. Acute symptomatic hyponatremia (120 mEq/L) is treated by 3% NaCl (513 mEq/L) over 4 hours. The requirement is calculated as follows:
   Na requirement in mEq/L = (Desired Na – Actual Na) × 0.6 × weight (kg).
   The desired Na is usually 125 mEq/L.
2. Clinical assessment should be done to look for vital signs, hydration status, fluid loss or underlying disease.
3. Laboratory assessment of serum electrolytes, urea, creatinine, glucose, serum osmolality and urine analysis for sodium, creatinine and osmolality should be done.
4. Serum sodium should be estimated and further correction if required is done over 24 to 48 hours. The possible course of events, cause and management is described in Table 2.4.5.

Management of other conditions like adrenal insufficiency with steroid, cardiac failure, cirrhosis.

True hyponatremia must be distinguished from factitious hyponatremia.

Factitious Hyponatremia

1. When nonpermeable substances (glucose, mannitol) are added to infusion, redistribution of water occurs from intra to extracellular space leading to hyponatremia.
2. Hyperlipidemia.

TABLE 2.4.5

Etiology and management of hyponatremia

Status	*Hypovolemic hyponatremia*	*Euvolemic hyponatremia*	*Hypervolemic hyponatremia*
Etiology	1. Renal loss	1. SIADH	1. Renal loss
	2. Extrarenal loss (gastrointestinal, sweat, third space)	2. Water intoxication 3. Drugs (mineralocorticoids)	2. Edematous state
Initial management	Isotonic saline	Water restriction	Salt and water restriction
Further management	5% dextrose with 0.45% saline	Frusemide, hypertonic saline	Hypertonic saline, dialysis

3. Hyperproteinemia.
4. Measured plasma osmolality is high.

The two syndromes presenting with hyponatremia are syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW).

Hypernatremic Dehydration

Normal serum sodium concentration is 138 to 145 mEq/L. Hypernatremia is present when serum sodium concentration exceeds 150 mEq/L. Serum sodium concentration more than 158 mEq/L is critical and may produce severe signs and symptoms.

Hypernatremia reflects a deficiency of water relative to total body sodium. It is actually a disorder of water balance rather than sodium.

Rapid increase in intravascular osmolality draws water from intracellular space to extracellular space causing intracellular dehydration. This altered water balance affects central nervous system also and results in brain shrinkage and blood vessel tear. It may lead to intracerebral bleeding, pulmonary edema and systemic arterial hypertension.

Hypovolemic Hypernatremia

Hypotonic fluid loss: This is the most common cause of hypernatremic dehydration in pediatric patient. It results from excessive loss of hypotonic fluid with inadequate water intake, like diarrhea with decreased water intake or vomiting. Osmotic diuresis in diabetes mellitus and osmotic diuretics like mannitol can induce hypernatremia secondary to excess hypotonic urine. In these conditions, the total body sodium is decreased but the total body water is reduced much more.

Water deficit: This is a condition of pure renal water loss secondary to diabetes incipidus, isolated renal water loss in excessive sweating, fever and hyperventilation.

Euvolemic Hypernatremia

This is the physiological state of sodium excess. It can occur as a result of feeding improperly mixed ORS solution or concentrated artificial feed to infants. Rarely does it occur due to excess intravenous sodium administration.

Clinical Features

1. Shock, doughy consistency of skin usually occurs with >10 percent dehydration.
2. Lethargy, irritability, coma and seizures.

3. Central nervous system dysfunction is severe and often leaves permanent sequelae.

Management

Initial Resuscitation

1. Severe dehydration and/or imminent shock should be corrected with isotonic fluid like NS or Ringer's lactate solution 20 mL/kg boluses till blood pressure becomes normal. Intravenous fluid containing sodium concentration at physiological level is ideal despite hypernatremia as it will drive water to the intracellular space. The aim is to correct intracellular dehydration.
2. Subsequently, 0.33 or 0.45 percent saline infusion is used till the child voids urine.
3. The aim is to correct dehydration over 48 hours.

Subsequent Management

Hourly fluid rate = Maintenance + Deficit/48

The fluid to be used is 0.33 or 0.25 percent saline in 2.5 percent Dextrose with adequate potassium. The sodium content of the fluid is adjusted, based on subsequent serum levels. The rate of fluid correction is more important than the sodium content.

If serum sodium falls >0.5 mEq/h, then intravenous fluid infusion rate is to be slowed down.

If serum sodium falls <0.5 mEq/h the infusion rate has to be increased.

Rate of fall in serum sodium level should ideally be 10 to 15 mEq/L/day. Too rapid correction of hydration and fall in serum sodium may cause cerebral edema, hypovolemia, pulmonary edema and central pontine myelinolysis.

1. Hypernatremic patients have predilection for hyperglycemia. Intravenous fluid after initial resuscitation should contain 2.5 percent dextrose, insulin is not required.
2. Hypocalcemia may require appropriate treatment.
3. Once the child voids urine potassium may be added at 40 mEq/L, it aids water entry into cells.
4. Diabetes incipidus may be treated accordingly.
5. Severe hypernatremia, serum sodium around 200 mEq/L may require peritoneal dialysis with high glucose (7.5%) and low sodium dialysate.

Hyperkalemia

Hyperkalemia is defined as serum potassium more than 5.5 mEq/L in children and more than 6.5 mEq/L in infants.

Etiology

1. Increased intake
2. Decreased renal excretion: Renal failure, potassium sparing dugs like spironolactone, ACE inhibitors, NSAIDs and mineralocorticoid deficiency
3. Increased movement into extracellular fluid like acidosis
4. Cell death or tissue necrosis.

Clinical Features

1. Muscle weakness
2. Cardiac arrhythmia.

ECG Changes

1. Tall T-wave
2. Long PR-interval, wide QRS
3. Absent P-wave, sinusoidal wave.

Management

1. Ensure sample not hemolyzed
2. Stop all exogenous potassium (IV, oral, drugs, blood product transfusion)
3. Cardiac monitor.

   Serum K >7 mEq/L or any hyperkalemia with ECG changes:

   i. Calcium (cardioprotective) with ECG monitoring—10 percent calcium gluconate 0.5 to 1 mL/kg/dose IV over 5 to 10 min. or 10 percent calcium chloride 0.2 mL/kg/dose IV over 5 to 10 min. It can be repeated up to 4 times or until serum calcium increases or ECG normalizes. Its effect lasts about 30 minutes.
   ii. $NaHCO_3$ 1 to 2 mmol/kg/dose IV over 30 minutes every 2 to 4 hourly. Do not mix with calcium in same IV line (results in precipitation).
   iii. Glucose 0.5 g/kg IV with insulin 0.1 unit/kg IV over 30 minutes. May be repeated in 30 to 60 min
   iv. Salbutamol nebulization
   v. Consider dialysis if above measures unsuccessful.

Hypokalemia

Hypokalemia is a state when serum potassium concentration is less than 2.5 mEq/L.

Etiology

1. Inadequate intake
2. Conditions when potassium is transferred into the cells—alkalosis, insulin.
3. Increased loss—diuretics, renal tubular acidosis, Bartter's syndrome, gastrointestinal loss, loss through skin like sweating or burns, excess steroid.

Clinical Features

Skeletal muscle weaknesses, abdominal distension with paralytic ileus are the most prominent features. Ployuria and polydipsia are secondary to loss of concentrating ability of the kidneys.

ECG Changes

ST segment depression, low and sometimes flat T-wave, in extreme cases U-wave. Increased risk of arrhythmia.

Management

1. ***Acute:*** Enteral replacement is preferable 1 to 4 mEq/24h in 2 to 4 divided doses. If parenteral replacement have to be given then 0.3 to 0.5 mEq/kg/h infusion by pump with cardiac monitoring. Central line is preferred. Serum potassium should be monitored closely.
2. ***Chronic:*** The daily dose to be calculated and replaced with potassium chloride or gluconate.

Hypocalcemia

Hypocalcemia is defined as serum calcium less than 2 mmol/L.

Clinical Features

Lethargy, poor feeding, vomiting, abdominal distension, twitching, tetany, seizures, apnea, stridor, laryngospasm.

ECG Changes

Prolonged Q-Tc interval (Normal <0.45 sec).

Management

Asymptomatic hypocalcemia: 100 mg/kg/d elemental calcium orally in divided every 6 to 8 hourly.

Symptomatic hypocalcemia:

i. 10 percent calcium gluconate 0.5 to 1.0 mL/kg/IV (diluted) over 5 to 10 min followed by 10 percent calcium gluconate 1 to 2 mL/kg/dose every 4 to 6 hourly bolus infusion diluted to 2 percent (Mix 10 mL of 10 percent calcium gluconate in 40 mL NS to obtain 2 percent solution). Or else continuous IV infusion 5 to 8 mL/kg/day of 10 percent calcium gluconate diluted to 2 percent. All patients on IV calcium should be on cardiac monitor.
ii. Integrity of the intravenous site should be ascertained before administering calcium through a peripheral vein.
iii. Adjust infusion rate q 4h based on plasma calcium level. Reduce infusion rate slowly once desired level reached then start oral calcium.
iv. Monitor IV site for extravasation burns and venous thrombosis.
v. Correct hypomagnesemia (Mg <0.6 mmol/L) because hypocalcemia does not respond until magnesium level is corrected.
vi. Magnesium sulfate 50 percent 0.2 mL/kg/dose IM/IV slowly.
vii. Consider starting vitamin D.

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2.5 BLOOD COMPONENT TRANSFUSION

Sudipta Sekhar Das

The available blood components are shown in Table 2.5.1.

TABLE 2.5.1
Blood components available

Products available	*Preparations*
1. Red blood cells (RBCs)	
Homologous packed RBC	Prepared from potential donors, are transfused most often. Promote oxygen delivery for patients with active bleeding or severe anemia
Leukocyte-poor RBC	Prepared by a variety of techniques to remove more than 99% of leukocytes. Reduce febrile reactions; Prevent HLA alloimmunization and CMV infection in potential transplant recipients or those requiring chronic platelet transfusions
Irradiated RBC	Reduces transfusion associated graft-versus-host disease (TAGVHD)
Washed RBC	Washing red blood cells in saline removes most plasma proteins and some leukocytes and platelets. Substitute for homologous RBC in patients sensitive to a plasma component; Avoid transfusion of anti-A and anti-B antibodies when O-negative blood is used in patients who are type A, B, or AB. Not often used today
Frozen RBC	RBCs frozen in liquid nitrogen with glycerol as a cryoprotective agent, can be stored for up to 10 years. Preserve autologous RBC; maintain store of rare blood types
Neocytes	Prepared by differential centrifugation or cell separators and have a longer circulating life span than standard red cells, but they are rarely used. Increase efficacy of individual transfusion for patients with transfusion-dependent anemia, thalassemia, etc.
Directed donor RBC	After screening and informed consent, may be substituted for volunteer RBC at patient request. Reduces multiple donor exposure in chronically transfused patients
Autologous RBC	May be collected preoperatively, by perioperative blood salvage, or by acute normovolemic hemodilution to decrease homologous red blood cell use

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2. Platelets	
Random donor platelets (RDP)	Obtained from whole blood
Platelet pheresis	Obtained by apheresis technology. Also known as single donor platelet (SDP). It has advantages over RDP
Leukocyte-poor platelets	Various techniques to prepare are available. It can remove >99% of leukocytes. It reduces febrile reactions and HLA alloimmunization and CMV transmission
Irradiated platelets	Reduces transfusion associated graft-versus-host disease
HLA matched platelets	Treat bleeding associated with thrombocytopenia in patients who are refractory to platelet transfusions due to HLA alloimmunization
3. Plasma and derivatives	
Fresh frozen plasma (FFP)	Prepared by separating plasma from whole blood and freezing it within 6 hours after collection at –18°C or colder. It can be stored for up to 1 year and is thawed prior to administration. It is very effective in coagulopathies
Fresh plasma (liquid plasma)	Same as FFP, except does not contain factors V and VIII. Used for preparation of plasma derivatives (e.g. immunoglobulin, cryoprecipitate, albumin, coagulation factor concentrates)
Cryoprecipitate-poor plasma	Supernatant plasma remaining after preparation of cryoprecipitate and contains adequate quantities of all coagulation factors except Fibrinogen, factors VIII and XIII, and vwf. Correct coagulation factor deficiencies other than VIII, XIII, fibrinogen, vwF; may be indicated for treatment of refractory TTP
Cryoprecipitate	Formed by thawing FFP at 4°C. This cryoprecipitate is separated from the supernatant plasma and resuspended in a small volume of plasma. It is then refrozen at –18°C and kept for up to 1 year
4. Granulocytes	
Leukapheresis	Granulocyte concentrates are prepared by automated leukopheresis from ABO-compatible donors stimulated several hours before collection with corticosteroids, G-CSF or both

Red Blood Cell

One unit of compatible red blood cells will increase the hemoglobin level by approximately 1g/dL or hematocrit by 3 percent. In neonates and older children, a dose of 10 to 15 mL/kg is usually given.

Indications

1. ***Chronic hypoproliferative anemia:*** Transfusions are rarely necessary for patients with chronic anemia who have hemoglobin concentrations above 7 g/dL unless significant cardiopulmonary disease is present.
2. ***Acute blood loss:*** Intraoperative blood loss may require red blood cell transfusion to maintain hemodynamic stability, and burn patients often require vigorous blood component support because of volume depletion through denuded body surfaces.
3. ***Hemolytic anemia:*** Patients with may require red blood cell transfusion until definitive therapy is effective.
4. ***Sickle cell anemia:*** Patients may require red blood cell transfusion for management of specific complications, including splenic sequestration and aplastic crises.
5. ***Perioperative transfusion:*** Transfusion is rarely indicated for patients undergoing noncardiac surgery who have hemoglobin values greater than 7–8 g/dL and no risk factors for myocardial ischemia.

Platelets

Whenever possible, ABO type specific platelets should be used in children. Platelets are always transfused in a dose of 5 to 10 mL/kg. Each platelet units of approximately 50 mL should contain a minimum of 3.5 to 4.5×10^{10} platelets.

Indications

1. Treatment of bleeding associated with thrombocytopenia or intrinsic platelet dysfunction.
2. Patients undergoing cardiopulmonary bypass may require platelet transfusions if excessive bleeding occurs because of thrombocytopenia and decreased platelet function induced by the bypass procedure.
3. Other surgical procedures in thrombocytopenic patients generally require prophylactic platelet transfusions to maintain adequate perioperative platelet counts for at least 3 days. More than 50,000/μL for most major procedures except neurologic, ophthalmic and cardiac procedures where platelet count of more than 100,000/μL is preferred.
4. Severely thrombocytopenic (e.g. <10,000/μL platelets) patients undergoing intensive chemotherapy for acute leukemia; the threshold for transfusion may be higher in the presence of fever, infection, or drugs that cause platelet dysfunction.

Platelet transfusions may be harmful in patients with TTP-HUS despite the presence of thrombocytopenia, presumably owing to accelerated thrombosis in vital organs. Patients with heparin-associated thrombocytopenia also may suffer increased thrombotic complications if platelets are transfused.

Plasma

The average child with multiple factor deficiencies requires 10–15 mL/kg of plasma acutely to control bleeding. The therapeutic products derived from plasma and their therapeutic uses are shown in Table 2.5.2.

Indications

1. Active bleeding due to deficiency of multiple coagulation factors, or risk of bleeding due to deficiency of multiple coagulation factors (DIC, liver disease).
2. Severe bleeding due to warfarin therapy, or urgent reversal of warfarin effect.
3. Massive transfusion with coagulopathic bleeding.
4. Bleeding or prophylaxis of bleeding for a known single coagulation factor deficiency for which no concentrate is available.
5. TTP-HUS, cryoglobulinemia, Goodpasture's syndrome, Guillain-Barré syndrome and post-transfusion purpura.

TABLE 2.5.2

Therapeutic products derived from plasma

Plasma derivative	*Therapeutic use*
Albumin (heat-treated)	Hypoalbuminemia in nephrotic syndrome
Plasma-derived factor VIII concentrate	Hemophilia A
Humate-P	Von Willebrand's disease
Prothrombin complex concentrate	Coagulation inhibitors, factor X and prothrombin deficiencies
Activated factor IX concentrates	Factor VIII inhibitors
Plasma-derived factor IX concentrate	Hemophilia B
Fibrinogen concentrate	Hypofibrinogenemia
Factor VII,XI,XIII concentrate	Factor VII,XI,XII deficiency
Antithrombin III concentrate	Thrombosis in antithrombin III deficiency
C1 esterase inhibitor concentrate	Angioedema
α1-Antitrypsin concentrate	Prevent lung damage in α1-AT deficiency
Protein C and S concentrate	Severe protein C or S deficiency
Intravenous immunoglobulin	Immunodeficiency states; immune cytopenias, Kawasaki syndrome, Guillain-Barré syndrome, dermatomyositis
Immune serum globulin	Passive immunization against hepatitis A measles, poliomyelitis, varicella, rubella

6. Plasma exchange may be of value in some patients with chronic inflammatory demyelinating polyneuropathy, cold agglutinin disease, autoimmune thrombocytopenia, rapidly progressive glomerulonephritis, and systemic vasculitis.

Plasma should not be administered for reversal of volume depletion or to counter nutritional deficiencies (except severe protein losing enteropathy in infants) because effective alternatives are available.

Complications of Transfusion

The infectious and noninfectious causes of complications of transfusion are shown in Tables 2.5.3 and 2.5.4, respectively.

TABLE 2.5.3
Infectious complications of transfusion therapy

Viruses	*Bacteria*	*Spirochetes*	*Parasites*	*Prion*
Hepatitis A, B, C Parvovirus B19 Cytomegalovirus Epstein-Barr virus HTLV 1 and 2 HIV West Nile virus	Environmental contaminants Donor transmitted Yersinia, Salmonella, Klebsiella Serratia Staphylococcus	Syphilis Lyme disease	Malaria Babesiosis Chagas' disease Toxoplasmosis	Variant Creutzfeldt-Jakob disease

TABLE 2.5.4
Nonhemolytic, noninfectious complications

Complication	*Clinical manifestations, pathogenesis, prevention and treatment strategies*
Febrile-nonhemolytic transfusion reaction (FNHTR)	Occurs in 0.5–3% of transfusion. Rigors or chills followed by fever during or shortly after transfusion due to prior sensitization to WBC or platelet antigens, or to pyrogenic cytokines released during storage. Prevent with antipyretics or leukocyte depletion of blood components before storage
Transfusion-related acute lung injury (TRALI)	Noncardiogenic pulmonary edema with fevers, chills, tachycardia, and diffuse pulmonary infiltrates shortly after transfusion, due to leukocyte incompatibility. Resolves in 1–4 days; rarely results in respiratory failure. Occurs in approximately 1:5000 transfusions

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Allergic reactions	Occurs in 1–3% of transfusions. Urticaria, pruritus, bronchospasm, or frank anaphylaxis due to recipient sensitization to a cellular or plasma element. If severe, evaluate recipient for IgA deficiency. Leukocyte depletion or washed red cells may be necessary for subsequent transfusions
Transfusion-associated circulatory overload (TACO)	Common following transfusion for chronic anemia or when patient has impaired cardiovascular reserve. Prevent by transfusing only when clearly indicated, using the minimum amount of blood required to reverse symptoms. Treat with oxygen, diuretics, and, rarely, phlebotomy (save units for reinfusion if necessary)
Dilutional effects	Transfusing with more than one blood volume or red blood cells with dilute platelets and coagulation factors. Replacement indicated only for clinical bleeding
Hypocalcemia	Due to citrate intoxication following massive transfusion. Treat only if symptomatic
Hyperkalemia	May occur in patients with preexisting renal insufficiency and hyperkalemia or in neonates. Use of fresh blood or washed red cells decreases potassium load for these patients
Hypothermia	After massive transfusion of refrigerated blood, hypothermia may cause cardiac arrhythmias. Refrigerated blood may accelerate hemolysis in patients with cold agglutinin disease. Prevent by warming blood
Transfusion associated Graft-versus-host disease	Immunocompetent donor T lymphocytes may engraft if the recipient is markedly immunosuppressed or if closely HLA-related. Symptoms and signs include high fever, maculopapular erythematous rash, hepatocellular damage, and pancytopenia, 2–30 days after transfusion. Usually fatal despite treatment with immunosuppressives. Prevent by irradiating all blood components with 2500 cGy for immunocompromised recipients or when donor is first-degree relative
Iron overload	Multiple transfusions in the absence of blood loss lead to excess accumulation of body iron with cirrhosis, heart failure, and endocrine organ failure. Prevent by decreasing total amount of red cells given, using alternatives whenever possible, using neocytes, and modifying diet to decrease iron absorption. Iron chelation indicated for patients with chronic transfusion dependence if prognosis is otherwise good
Post-transfusion purpura	Acute severe thrombocytopenia about 1 week after transfusion due to alloantibodies to donor platelet antigen, usually human platelet antigen 1A (HPA 1A). Self-limited, but treatment with steroids, high-dose IgG, plasmapheresis, or exchange transfusion recommended to prevent CNS hemorrhage. Platelet transfusions are ineffective even with compatible platelets

Nonhemolytic, noninfectious transfusion reactions account for more than 90 percent of adverse effects of transfusions and occur in approximately 7 percent of recipients of blood components.

Miscellaneous

Increased supply of complement may accelerate hemolysis in paroxysmal nocturnal hemoglobinuria or make angioedema worse in patient with C1 esterase inhibitor deficiency. Increased blood viscosity may occur in patients with Waldenström's macroglobulinemia, polycythemia, or leukemia with high WBC count.

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3

Sedation, Analgesia, Intubation and Procedures

Jayanta Bandyopadhyay, Mahasweta Chaudhuri

3.1 BASICS OF INTUBATION

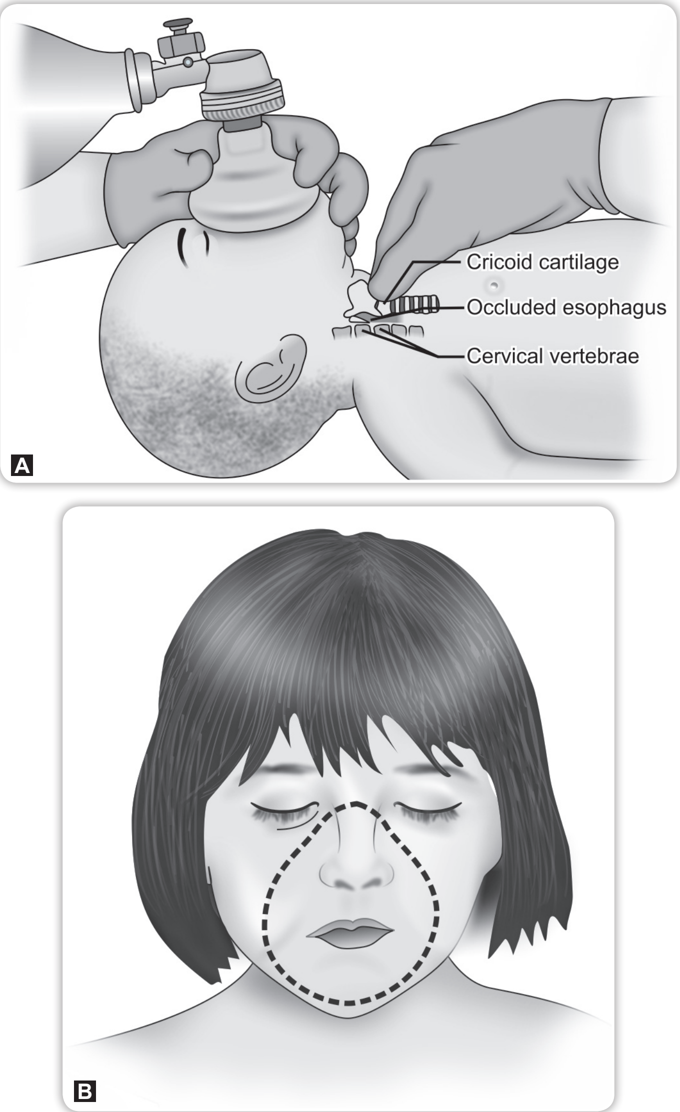
Jayanta Bandyopadhyay, Mahasweta Chaudhuri

Intubation is the definitive advanced life support technique for airway management. The purpose is to either secure the airway or to ensure adequate ventilation and oxygenation. The pediatric airway is particularly challenging due to the small patient size, differences in anatomy, and relatively high oxygen demand in pediatric patients. The characteristics of pediatric airway and the associated implications during intubation is summarized in Table 3.1.1. Figures 3.1.1A and B shows technique of bag and mask ventilation.

TABLE 3.1.1

Pediatric airway and its implications during intubation

Characteristics	*Implications*
Infant epiglottis is omega (Ω) shaped and angled away from the axis of trachea	This anatomy necessitates use of straight blade laryngoscope
Relatively larger tongue, which can obstruct airway	Commonest cause of airway obstruction May necessitate better head positioning
Larynx is more rostral and anterior	Difficult to visualize the cord, a shoulder roll may be needed, may have to visualize the cord at 45° angle or greater
Narrowest part of pediatric airway is uncuffed tube, which are used in children up to 8 years, cricoid cartilage, however, present studies show cuffed tube may be used safely if needed	

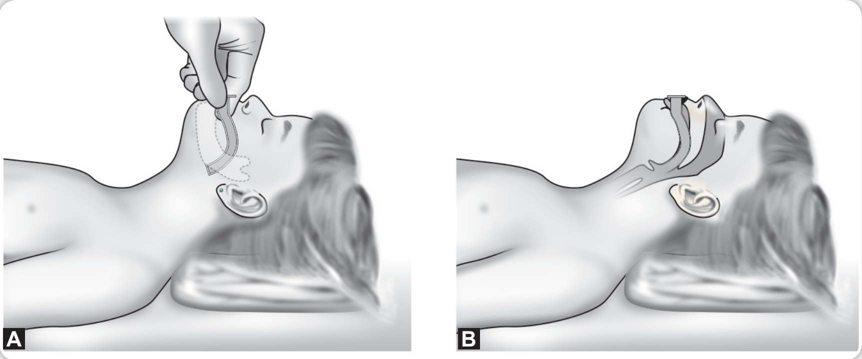


Figures 3.1.1A and B Technique of bag and mask ventilation

Indications for Intubation

Oropharyngeal airway and position is shown in Figures 3.1.2A and B.

1. Provide airway in functional or anatomic airway obstruction (chest trauma with flail chest, maxillofacial injury, epiglottitis, peritonsillar abscess).
2. Protect airway in patients with loss of cough and gag reflexes and copious tracheal secretions as a result of inadequate CNS control or neuromuscular diseases (apnea, head injury, GB syndrome and poliomyelitis).



Figures 3.1.2A and B Oropharyngeal airway—proper size and position

3. Instituting positive pressure ventilation (pneumonia, ARDS, pulmonary edema, bronchiolitis).
4. Circulatory disorder (cardiac arrest, shock, pulmonary artery hypertension).
5. Elective—Before any major and/or extensive surgery, till hemodynamically stable.

Equipment

1. Suction apparatus and tracheal suction catheter
2. Oxygen
3. Appropriate endotracheal (ET) tube—different sizes
4. Bag valve mask device
5. Laryngoscope, preferable straight blade
6. Tincture benzoin, tape
7. Medications
8. Monitoring equipment
9. Experienced medical and paramedical assistants.

Selection of Laryngoscope Blade

Miller blade is preferred for infants and younger children. It facilitates lifting of the epiglottis and exposing the glottic opening. Care must be taken to avoid using the blade as a fulcrum with pressure on the teeth and gums. Macintosh blades are generally used in older children.

Endotracheal Tube Size

The appropriate ET tube, laryngoscope blade and suction catheter details are represented in Table 3.1.2.

TABLE 3.1.2

ET tube, laryngoscope blade and suction catheter details

Body weight/age	*Tube size (mm)*	*Depth of insertion (cm)*	*Laryngoscope blade (Miller)*	*Laryngoscope blade (Macintosh)*	*Tracheal suction catheter (F)*
1500–3000 gm	3.0	7–9	0	-	5
Neonate to 6 months	3.5	10	0	-	6
6–18 months	4.0	11	1	-	6–8
2 years	4.5	12	1	2	8
>2 years	Age (years)/	Age (years)/	2–6 years = 1	2	10–12
(8–10 years)	4 + 4	2+ 12	6–10 years = 2	2–3	
			>11 years = 2-3	3–4	
Adult	8–8.5 (Men)	21–23	3	4	14
	7.5–8 (Women)				

Nasal intubation should not be tried in the following:

1. Need for immediate airway access
2. Fracture at the base of skull, faciomaxillary trauma
3. Coagulopathy.

Pharmacological Control Over Intubation

1. ***Without any premedication:*** Done in a moribund patient with impending cardiorespiratory arrest where a delay to paralyze may be detrimental. Access may be difficult due to struggle, bite and may cause trauma to airway, laryngospasm, deteriorating vital signs during the procedure. This is the commonest situation in emergency room.
2. ***With sedation:*** This should be attempted by the most experienced hand. Sedation may be used where patient has a low or moderate oxygen requirement (using fentanyl and midazolam followed by suxamethonium and then vecuronium). Avoid using ketamine if suspecting raised intracranial tension. But intubation using sedation runs the risk of compromised airway reflexes and hence chance of aspiration.

3. ***Anesthetized:*** Done in totally controlled and elective situation like prior to major surgery (using thiopentone, propofol/ketamine according to individual choice and case to case basis).

Preparing for the Procedure

While intubating a patient, there are certain basic essentials that must be present to ensure a safe intubation. They can be remembered by the mnemonic **SALT**.

Suction: It is the first and an extremely important step in the whole procedure. Often patients may have secretions in the pharynx that make visualization of the vocal cords difficult.

Airway: Oxygen source with proper delivery mechanism (bag and mask) must be available. Medication and monitoring equipment should be ready.

Laryngoscope: Proper size with good batteries should be ready for placing an endotracheal tube.

Tube: Endotracheal tubes of appropriate sizes.

Positioning the Patient

1. Child above 2 years: A folded towel to be placed under the occiput to align the pharyngeal and tracheal axis.
2. Child below 2 years: A folded sheet to be placed under shoulder to align airway.

Procedure

1. The child should be assessed, vital signs, level of consciousness and oxygen saturation should be recorded. Gastric content should be aspirated to avoid aspiration during intubation
2. Preoxygenate and ventilate with 100 percent O_2 using bag-valve mask. Premedicate as per requirement
3. Airway is visualized directly using laryngoscope
4. When vocal cords have been visualized, the endotracheal tube is passed through the cords
5. Ventilation is initiated and tube placement is confirmed
6. Proper tube placement should be confirmed by the following:
    i. Auscultate to confirm equal breath sounds in axilla and no sound over gastric area
    ii. Water vapor in the tracheal tube during exhalation
    iii. Pulse oximetry to confirm O_2 saturation.

Endotracheal tube should be secured. Head immobilization should be done to prevent tube dislodgement. Chest X-ray should be taken to check tube placement, and also obtain arterial blood gas measurements to assess the adequacy of ventilation.

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4

Neonatal Emergencies

Jaydeep Choudhury, Jayanta Bandyopadhyay

4.1 NEONATAL EMERGENCIES

Jaydeep Choudhury

Identifying a sick neonate in an emergency situation is always a challenge. A high index of suspicion is the key when a neonate presents with altered mental status. The following causes as per the mnemonic THE V SEPSIS may be remembered as a basic guide.

Causes of a neonate presenting altered mental status: THE V SEPSIS

T – Trauma
H – Heart diseases
E – Electrolyte imbalance
V – Volume imbalance (hypovolemia, inappropriately prepared formula)
S – Seizures
E – Endocrine (congenital adrenal hyperplasia, thyrotoxicosis)
P – Poison and toxins
S – Surgical (volvulus, intussusception, necrotizing enterocolitis)
I – Inborn errors of metabolism
S – Sepsis

Severe Hypothermia

Ideally, a rectal temperature should be measured and the thermometer should be kept for 3 minutes in neonates, 2 minutes in infants and children.

Temperature Range

Normal temperature:	36.5–37.5°C
Cold stress:	36–36.5°C

Moderate hypothermia: 32–36°C
Severe hypothermia: <32°C

Management

Moderate Hypothermia

1. Skin to skin contact (Kangaroo method in neonate)
2. Frequent monitoring, every 15 minutes
3. Frequent feeding.

Severe Hypothermia

It is a life threatening condition and needs immediate care. The management comprises of the following:

1. Radiant warmer which can provide extra heat and cause rapid rewarming. Temperature should be rapidly raised to 34^{o}C and then slowly
2. Room heater
3. Oxygen
4. Dextrose 10 percent IV 2 to 4 mL/kg bolus, blood glucose should be checked regularly
5. Intravenous fluid infusion.

Seizures

It is very important to identify subtle seizures like abnormal eye movements, lip smacking, abnormal tongue movements, repetitive chewing, drooling, yawning, pedalling, apnea, focal or multifocal clonic, tonic posturing of limbs and myoclonic seizures as typical seizures are often not observed in neonates.

Management

1. Stabilization: T, A, B, C.
2. An intravenous line should be established. Blood sample should be collected for sugar, calcium, magnesium, electrolytes, blood cell count and CRP. The following management is continued one step at a time till seizure is controlled, throughout taking care of the vital functions.
3. Hypoglycemia (≤40 mg/dL) should be corrected immediately. Intravenous injection 10 percent dextrose 2 to 4 mL/kg over 3 minutes. If the seizure gets controlled then intravenous infusion is started with dextrose at the rate of 6 to 8 mg/kg/min.
4. Lorazepam 0.1 mg/kg IV is the initial drug of choice. Lorazepam has got smaller volume of distribution and longer half-life than diazepam. Diazepam 0.1 to 0.3 mg/kg may also be given.

5. Phenobarbitone is the next choice, 15 to 20 mg/kg is given slow intravenous. If the seizure is not controlled after 15 minutes, a second loading dose 10 to 20 mg/kg can be given. Maximum total phenobarbitone loading dose is 50 to 60 mg/kg. Maintenance dose is 5 mg/kg in two divided doses.
6. Phenytoin 15 to 25 mg/kg loading dose diluted in normal saline may be given slow intravenous over 15 minutes. Maintenance dose is 5 mg/kg in two divided doses.
7. If hypocalcemia is present (≤7 mg/dL), calcium gluconate 10 percent solution, 2 mL/kg is injected slowly over 5 to 10 minutes under cardiac monitor.
8. Hyponatremia (≤125 mg/dL) should be corrected with 3 percent saline. The replacement calculation is as follows.
   (125 – Serum Na) × weight × 0.6 mEq of Na^+ (1 mL = 0.5 mEq), slowly over 4 hours.

Hypoglycemia

By definition blood glucose <30 mg/dL is hypoglycemia. But neonates with blood glucose between 30 and 40 mg/dL may be symptomatic. Thus any blood glucose less than 40 mg/dL should be treated.

Management

1. Intravenous injection 10 percent dextrose 2 to 4 mL/kg over 3 minutes.
2. Followed by intravenous infusion started at the rate of 6 to 8 mg/kg/min.
3. If the seizure is not controlled infusion may be hiked to 12 mg/kg/min. The maximum allowed tonicity by a peripheral line is 12 percent. A central venous line is necessary to give glucose at 15 to 20 percent.
4. If the seizure gets controlled, the rate of infusion is continued for next 24 hours.
5. Hydrocortisone—5 mg/kg injection twice daily is given, if the seizure is controlled, the same dose may be continued for 3 to 5 days.
6. Glucagon—0.1 mg/kg, maximum 1 mg may be given intramuscularly.
7. Diazoxide—5 to 15 mg/kg/day in 3 divided doses oral.

Neonatal Hemorrhage

Hemorrhage in neonates is always life-threatening. The hemorrhage may be either internal or external.

Management

1. Stabilization of the neonate is most important. Blood from mouth and nose should be sucked and cleaned to prevent aspiration. It also keeps the

airway patent. Oxygen may be administered. An intravenous fluid should be started.

2. Blood sample to be collected for Hb, PCV, cell type and count, platelet count, prothrombin time, partial thromboplastin time, sugar and CRP.
3. Vitamin K—2 mg intravenous or intramuscular.
4. Ranitidine—1 mg/kg stat, followed by 12 hourly.
5. Packed cell transfusion 10 mL/kg in case of severe hemorrhage.
6. At this stage further assessment has to be done and laboratory reports analyzed. The following are the possibilities:
    i. Normal platelet count, elevated prothrombin time: Vitamin K injection to be repeated and prothrombin time repeated after 12 hours.
    ii. Low platelet count sick—child with positive tests for sepsis then the condition has to be treated according to the etiology like congenital infection, disseminated intravascular coagulation or congenital heart disease.
    iii. Low platelet count—mother's platelet count has to be checked.
    iv. If mother's platelet count is normal—congenital anomalies like giant haemangioma has to be looked for.
    v. If mother has low platelet count—maternal ITP, SLE, drugs like sulphonamide or quinine intake. Baby born to mother suffering from ITP or SLE should be given intravenous immunoglobulin 400 mg/kg/day for 2 to 5 days and prednisolone 2 mg/kg/day.

Neonatal Apnea

Preterm infants are more prone to apnea due to immaturity in all or any of the components of respiratory control—central or peripheral.

Apnea if present for 20 sec produces fall in PaO_2. Cyanosis develops after 30 sec and after 45 sec there is bradycardia, pallor, hypotension and unresponsiveness.

Management

1. Temperature maintenance, control of sepsis if any, normoglycemia and control of polycythemia are important aspect as hypothermia may trigger apnea.
2. A particular head neck posture may also trigger apnea, which should be avoided.
3. All infants less than 34 weeks of gestational age should be monitored for apneic spells for at least the first week of life. Along with impedance apnea monitors heart rate should also be monitored.
4. Most apneic spells in premature infants respond to tactile stimulation.
5. Unresponsive patients should be ventilated during spell with bag and mask with a FiO_2 under 0.4.

6. Pharmacotherapy: Indications are two or more episodes in an 8 hour interval that require physical stimulation or a single episode that requires bag and mask ventilation. The following drugs may be used:
   - Caffeine 10 mg/kg loading dose oral or intravenous followed by 2.5 mg/kg/day.
   - Aminophylline 6–8 mg/kg loading dose intravenous followed by 2 to 6 mg/kg/day in two divided doses.
   - Theophylline 5.5–7 mg/kg loading dose orally followed by 2.5 to 8 mg/kg/day in 3 divided doses.
   - The drugs may be discontinued at 37 weeks postnatal age.
7. Nasal CPAP: A pressure of 3–4 cm H_2O reduces mixed and obstructive apnea.
8. Ventilation: Indications are total respiratory failure, failure of pharmacotherapy and CPAP.

Air Leak

It is usually an outcome of mechanical ventilation particularly in the background of meconium aspiration syndrome.

Management

1. *Conservative therapy:* It is the treatment of choice in infants with noncontinuous air leak, no underlying lung disease, no distress and not on mechanical ventilator. Close observation is necessary.
2. *Needle aspiration:* It may be employed as diagnostic and therapeutic procedure in distressed neonate. A 23 or 25 gauge needle with a 3-way stop cock is inserted through the second intercostals space through the midclavicular line grazing the upper border of the rib. A continuous suction should be applied as the needle is inserted. A rapid flow of air will occur when the pneumothorax is entered. Once airflow ceases, needle should be withdrawn.
3. *Chest tube insertion:* It is necessary in neonate with continuing air leak. No. 10 or 12 French chest tubes are most commonly used. A small incision should be made along the 6th rib in the anterior axillary line. The tube should be inserted bluntly guided by a hemostat through the 3rd or 4th intercostals space. The tube should be secured well and connected to a water seal. Chest X-ray should be frequently taken to see the progress. The tube is removed when the lung disease has improved and the tube has drained no air for 24 to 48 hours.

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4.2 BLEEDING NEONATE

Jaydeep Choudhury, Jayanta Bandyopadhyay

Both term and preterm neonates may present with bleeding due to various causes. Quick assessment is very important as these children may quickly go into shock.

Baseline Investigations

1. Complete blood count, RBC morphology.
2. Platelet count.
3. Prothrombin time (PT), Partial thromboplastin time (PTT), thrombin time (TT) and fibrinogen.
4. Sepsis screen—blood culture, CRP.

Management

Supportive Care

1. Stabilization—A, B, C and thermoneutral environment
2. Monitoring for shock and appropriate fluid resuscitation
3. Injection vitamin K, 1 mg IV.

Neonate in Shock

1. Transfusion of O negative blood, 10 to 15 mL/kg over 5 to 15 minutes
2. The above can be repeated once if the neonate is still in shock
3. Monitoring BP: Aim is to keep above 40 mm Hg
4. Transfer to NICU for management.

Neonate not in Shock

Transfusion of matched blood.

Specific Treatment

1. Vitamin K injections
2. Fresh frozen plasma
3. Platelet transfusion
4. Whole blood transfusion.

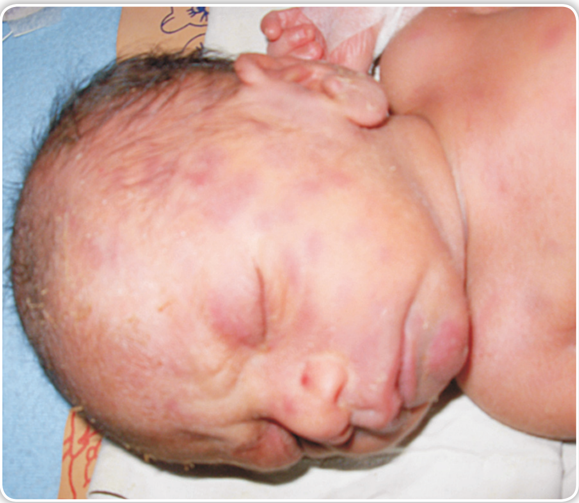
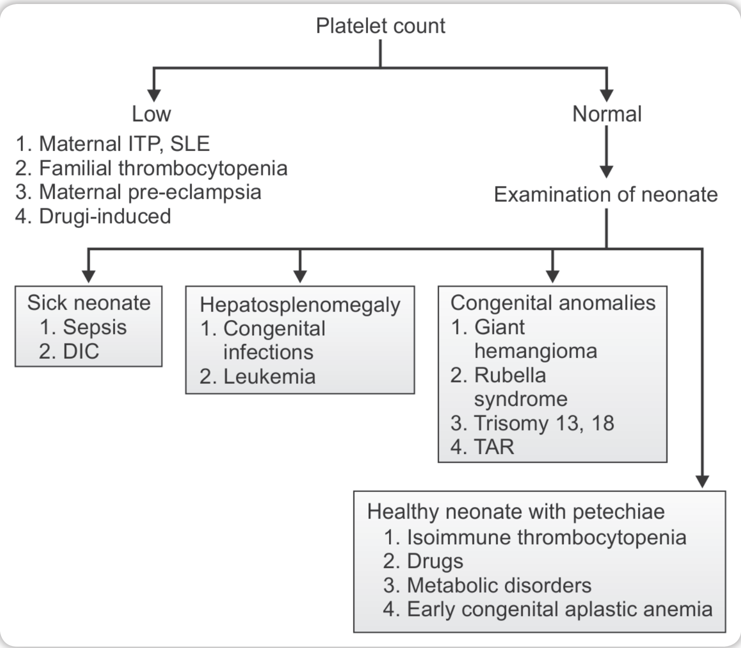


Figure 4.2.1 A child with neonatal purpura

Flow chart 4.2.1 Diagnostic approach to bleeding disorder in neonates



A child with neonatal purpura and the diagnostic approach to a bleeding neonate are shown in Figure 4.2.1 and Flow chart 4.2.1, respectively.

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4.3 NEONATAL COLLAPSE

Jayanta Bandyopadhyay

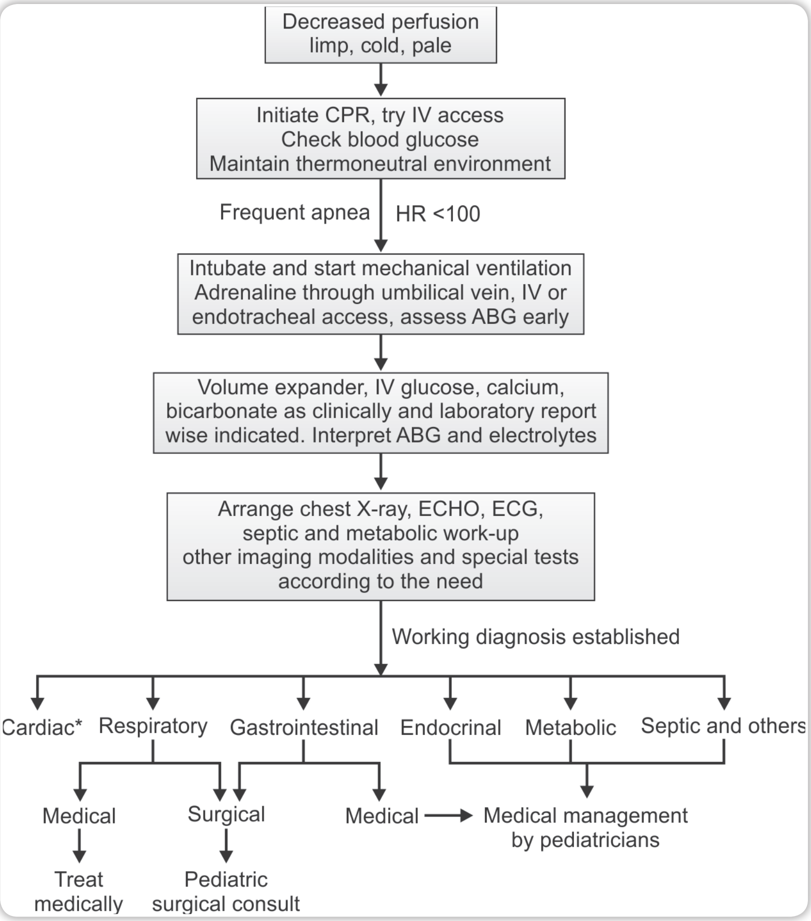
This is a state where a neonate in a very short span of time or in other words almost suddenly becomes pale, limp, ashen gray with cold extremities, absent peripheral pulses and apneic or has shallow respiration and rapidly nosedives to cardiorespiratory arrest.

Management

1. Initiate CPR, check blood glucose.
2. Maintain a thermoneutral environment to minimize oxygen and glucose requirement.
3. Intubate if primary apnea, HR <100/min after initial airway maneuvers and start mechanical ventilation. Assess ABG.
4. Tracheal adrenaline or injection via umbilical vein.
5. Volume expansion with normal saline, fresh frozen plasma, packed cell (as condition necessitates) 10 mL/kg and repeat according to the clinical condition.
6. Bicarbonate for acidosis, diluted and slowly.
7. Calcium gluconate 2 mL/kg of 10 percent solution slowly IV both for hypocalcemia and more so for its positive inotropic action.
8. IV glucose injection for hypoglycemia.
9. Prostaglandin E1 (PGE1)—if proved duct dependent circulation.
10. Inotropes—dopamine 5 to 10 µg/kg/min.
11. Add dobutamine if low cardiac output but normal or increased peripheral vascular resistance.
12. Neonate with cardiogenic shock—isoproterenol (0.1–0.5 µg/kg/min) and digoxin with be helpful.
13. Milrinone and inamrinone (3 mg bolus and then 5–20 mg/kg/min) reduce afterload by their vasodilator effect.
14. Antibiotics according to institutional protocol for sepsis, NEC and meningitis.
15. Central venous pressure and arterial blood pressure should be monitored to guide therapy though technically may be difficult in newborns.
16. Naloxone for opiate induced respiratory depression.
17. During salt loosing period or when vomiting is present in congenital adrenal hyperplasia, apart from fluid rescitation, hydrocortisone 50 mg/m^2 stat followed by 100 mg/m^2/day every 6 hourly IV till stabilizes then 20 mg/ m^2/day and add fludrocortisone 0.15 mg/m^2/day. Maintain hydrocortisone at 9.5–15 mg/m^2/day as it causes least growth inhibition compared to dexamethasone and prednisolone. Continue fludrocortisone 0.15 mg/m^2/day lifelong.

Management algorithm for neonatal collapse is shown in Flow chart 4.3.1.

Flow chart 4.3.1 Management algorithm for neonatal collapse


Decreased perfusion
limp, cold, pale
Initiate CPR, try IV access
Check blood glucose
Maintain thermoneutral environment
Frequent apnea
HR <100
Intubate and start mechanical ventilation
Adrenaline through umbilical vein, IV or endotracheal access, assess ABG early
Volume expander, IV glucose, calcium, bicarbonate as clinically and laboratory report wise indicated. Interpret ABG and electrolytes
Arrange chest X-ray, ECHO, ECG, septic and metabolic work-up other imaging modalities and special tests according to the need
Working diagnosis established
Cardiac*
Respiratory
Gastrointestinal
Endocrinal
Metabolic
Septic and others
Medical
Surgical
Medical
Medical management by pediatricians
Treat medically
Pediatric surgical consult

*Cardiac causes
→ Duct dependant circulation—start PGE1, consult pediatric cardiologist
→ Severe valvular stenosis and COA—consult pediatric cardiologist for balloon dilatation of valve and balloon angioplasty respectively
→ TOF physiology—consult surgeon for BT shunt
→ Other medical causes—specific treatment as per guidelines.

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5

Respiratory Emergencies

Jayanta Bandyopadhyay, Jaydeep Choudhury, Saheli Misra

5.1 RESPIRATORY DISTRESS AND NOISY BREATHING

Jayanta Bandyopadhyay, Jaydeep Choudhury

Acute respiratory distress is a potential life-threatening condition. Respiratory distress may be due to upper airway, lower airway or sometimes due to a nonrespiratory cause where there is decreased lung compliance.

Nonrespiratory Causes of Respiratory Distress

Cardiac condition should always be assessed in a child with respiratory distress. Heart failure may present with distress. Acidosis may produce respiratory distress. Renal failure, liver disease also cause distress. Drug history may be useful as overdose may cause respiratory distress.

Noisy Breathing

Noisy breathing in children is usually due to the various causes of upper airway obstruction.

Approach to Noisy Breathing

Diagnosis and management of various emergency situations of noisy breathing in children is discussed in other sections. Few important points for noisy breathing are as follows:

1. Focused and detailed history
2. Examination of nose—septum, adenoid, foreign body

TABLE 5.1.1

Differences between croup, epiglottitis and foreign body aspiration

Parameters	*Croup*	*Epiglottitis*	*Foreign body*
Age	6 – 18 months	2 – 5 years	> 6 months
Onset	Gradual, over 24 – 48 hours	Sudden	Sudden
Prodromal features	Present	Nil	Nil
Temperature	May be mildly elevated	High	Normal
Stridor	High pitched loud	Low pitched soft	Variable, depends on location of foreign body
Barking cough	Present	No cough	Variable
Hoarse voice	Present	Voiceless	Variable
General appearance	Noisy and playful	Anxious, sits still	Depends on degree of obstruction

3. Throat and larynx—tonsils, diphtheritic patch, foreign body
4. Pharynx and epiglottis
5. Lymphadenopathy
6. ENT consultation at the earliest.

   Differentiating features between croup, epiglottitis and foreign body aspiration.

   The differences are shown in Table 5.1.1.

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5.2 CROUP

Jayanta Bandyopadhyay, Jaydeep Choudhury

Viral laryngotracheobronchitis or croup is the commonest cause of acute upper airway obstruction.

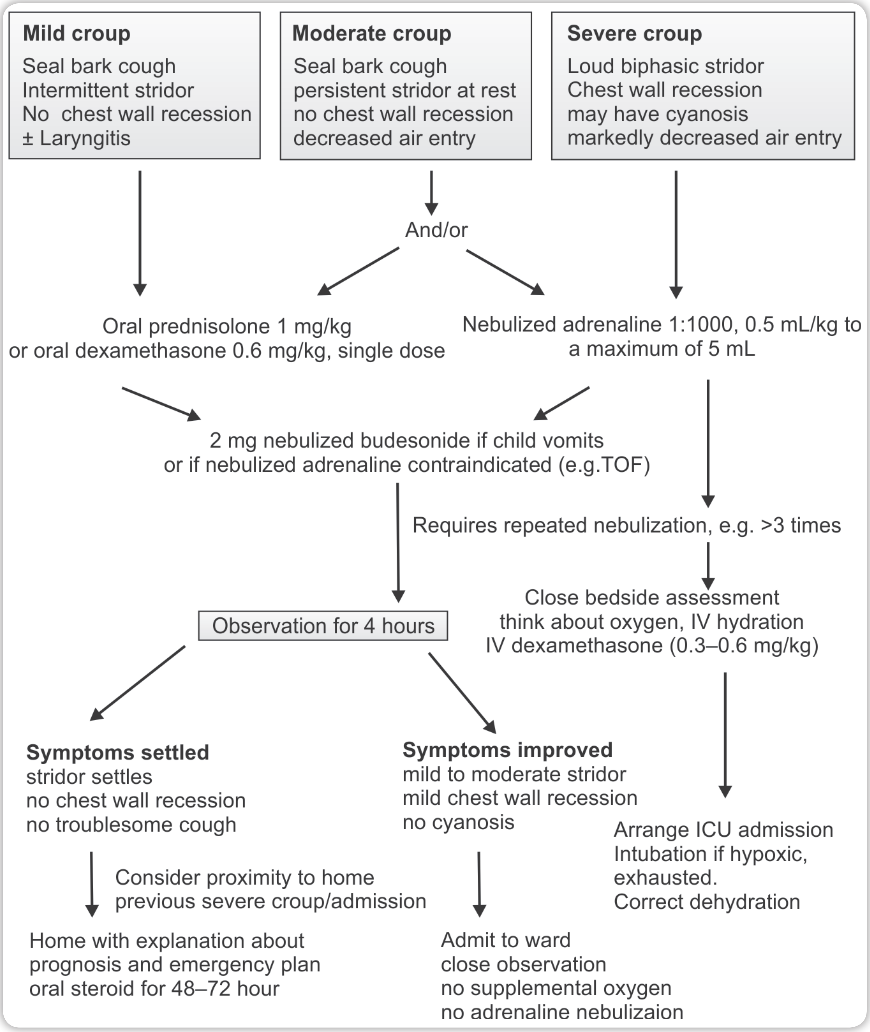
Management

1. Accompanying stridor often begins at night awakening infants and toddlers from sleep.
2. No role of antibiotics in viral croup.
3. Blood gases are usually not necessary (may precipitate still worse obstruction) and little to be gained from chest and neck X-rays in typical cases (shifting to radiology room may be risky idea).
4. Pulse oxymetry is on the other hand reliable noninvasive way of monitoring.
5. Assessment of severity using Westley Clinical Scoring System in emergency department, as shown in Table 5.2.1 is very useful to plan for staying at observation unit, hospital admission, ICU admission.
6. Agitation, exhaustion, cyanosis, severe retractions, markedly decreased air entry are signs of impending danger of airway obstruction.

TABLE 5.2.1

Clinical scoring system. By Westley CR, Cotton EK, Brooks JG

Level of consciousness	Normal (including sleep)	0
	Disoriented	5
Cyanosis	None	0
	Cyanosis at agitation	4
	Cyanosis at rest	5
Stridor	None	0
	When agitated	1
	At rest	2
Air entry	Normal	0
	Decreased	1
	Markedly decreased	2
Retraction	None	0
	Mild	1
	Moderate	2
	Severe	3


Mild croup
Seal bark cough
Intermittent stridor
No chest wall recession
± Laryngitis
Moderate croup
Seal bark cough
persistent stridor at rest
no chest wall recession
decreased air entry
Severe croup
Loud biphasic stridor
Chest wall recession
may have cyanosis
markedly decreased air entry
And/or
Oral prednisolone 1 mg/kg
or oral dexamethasone 0.6 mg/kg, single dose
Nebulized adrenaline 1:1000, 0.5 mL/kg to
a maximum of 5 mL
2 mg nebulized budesonide if child vomits
or if nebulized adrenaline contraindicated (e.g.TOF)
Requires repeated nebulization, e.g. >3 times
Observation for 4 hours
Close bedside assessment
think about oxygen, IV hydration
IV dexamethasone (0.3–0.6 mg/kg)
Symptoms settled
stridor settles
no chest wall recession
no troublesome cough
Symptoms improved
mild to moderate stridor
mild chest wall recession
no cyanosis
Arrange ICU admission
Intubation if hypoxic,
exhausted.
Correct dehydration
Consider proximity to home
previous severe croup/admission
Home with explanation about
prognosis and emergency plan
oral steroid for 48–72 hour
Admit to ward
close observation
no supplemental oxygen
no adrenaline nebulizaion

Flow chart 5.2.1 Croup management plan

7. Endotracheal intubation if planned must be performed by a specialist preferably an anesthesiologist because of the risk of laryngospasm precipitated by instrumentation.

   The step wise management of croup is shown in Flow chart 5.2.1.

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5.3 ACUTE EPIGLOTTITIS

Saheli Misra, Jaydeep Choudhury

Acute epiglottitis is a life-threatening bacterial infection of the epiglottitis and aryepiglottic folds.

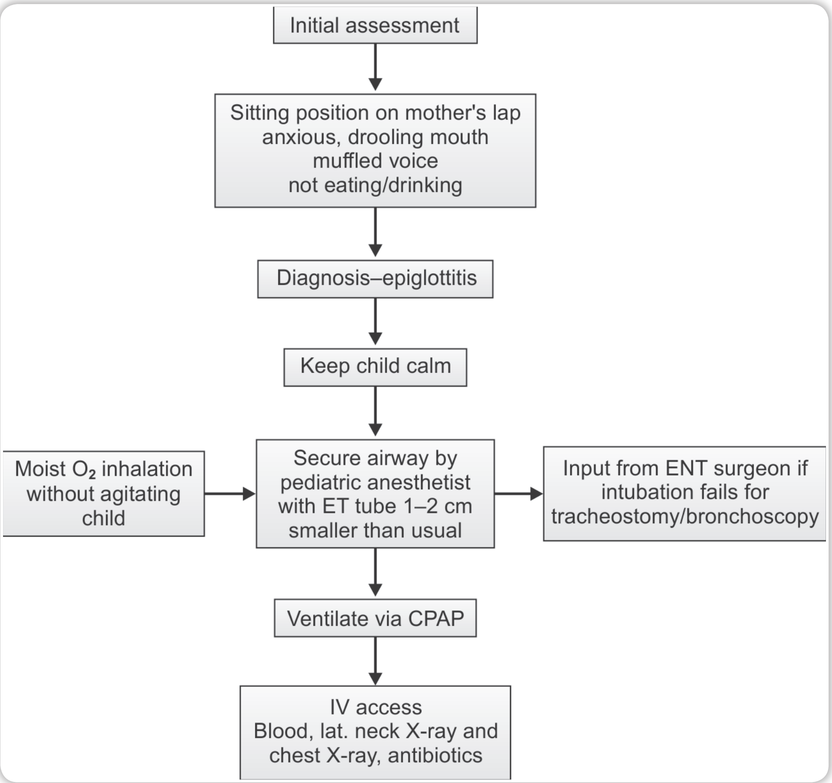
Management

The algorithm for management of acute epiglottitis is shown in Flow chart 5.3.1.

Recommended Antibiotics

1. Cefotaxime 200 mg/kg/day IV divided every 8 hours, or ceftriaxone 100 mg/kg/day IV divided every 12 to 24 hours. First dose stat.

Flow chart 5.3.1 Management of acute epiglottitis


Initial assessment
Sitting position on mother's lap anxious, drooling mouth muffled voice not eating/drinking
Diagnosis–epiglottitis
Keep child calm
Moist O_2 inhalation without agitating child
Secure airway by pediatric anesthetist with ET tube 1–2 cm smaller than usual
Input from ENT surgeon if intubation fails for tracheostomy/bronchoscopy
Ventilate via CPAP
IV access Blood, lat. neck X-ray and chest X-ray, antibiotics

2. If bacterial tracheitis is suspected add better gram-negative coverage. Respiratory isolation for 24 hours after initiation of antibiotics should be ordered.

Indications and guidelines for rifampicin chemoprophylaxis for contacts of index cases of invasive *Haemophilus influenzae* type b (Hib) disease

Dose of rifampicin—20 mg/kg orally once daily for 4 days, maximum 600 mg/day.

Chemoprophylaxis Recommended

1. All household contacts (except pregnant women), irrespective of age, with at least 1 contact younger than 4 years of age who is unimmunized or incompletely immunized. The index patient also should receive chemoprophylaxis.
2. All members of a household with a child younger than 12 months of age, even if the primary series has been given.
3. All occupants of a household with an immunocompromised child, irrespective of the child's Hib immunization status.
4. Nursery and child care center contacts, irrespective of age, when 2 or more cases of invasive disease have occurred within 60 days.
5. Index case, if treated with regimens other than cefotaxime or ceftriaxone. Chemoprophylaxis usually is provided just before discharge.

Chemoprophylaxis not Recommended

1. Occupants of households with no children younger than 4 years of age other than the index patient.
2. Occupants of households when all household contacts younger than 48 months of age have completed their Hib immunization series.
3. Nursery and child care center contacts of one index case, especially those older than 2 years of age.
4. Pregnant women.

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5.4 BRONCHIOLITIS

Jayanta Bandyopadhyay, Jaydeep Choudhury

Bronchiolitis is inflammation of the lower respiratory tract due to an infection, commonly viral. Bronchiolitis assessment tool (BAT) is shown in Table 5.4.1.

The illness peaks at 2 to 3 days and improves by 5 to 7 days. Cough may persist for several weeks.

Management

1. Small frequent feeds. In moderate and severe cases nasogastric feeds may be needed. In severe respiratory distress it may be safest not to feed and give IV fluids.
2. Saline nasal drops may facilitate feeding if there is a problem with nasal obstruction.
3. Oxygen should be administered as necessary to maintain adequate saturation (>92%). O_2 is usually administered via a nasal cannula.
4. Infants who are not maintaining satisfactory oxygenation with nasal O_2 or who are having apneic episodes require PICU assessment.
5. Most children with bronchiolitis do not benefit from bronchodilators and there is some evidence that nebulized bronchodilators can cause babies to deteriorate. Some older infants may benefit from bronchodilators.
6. Antibiotics are of no benefit in bronchiolitis. A small number of children with bronchiolitis may develop secondary bacterial chest infection. This should be considered if there is a deterioration associated with a fever.
7. Steroids are not indicated for bronchiolitis.
8. Ribavirin is indicated in immunocompromised patients.

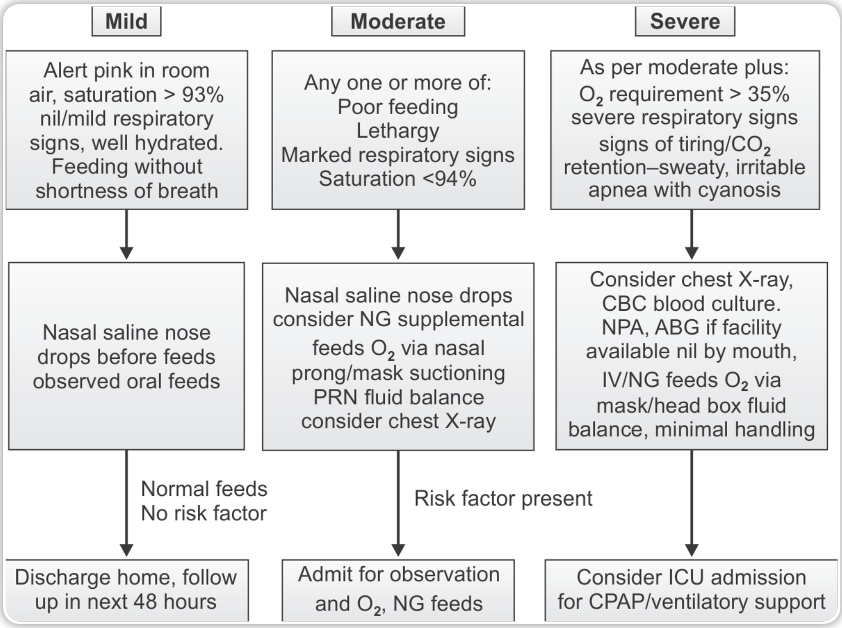
The management plan of bronchiolitis is shown in Flow chart 5.4.1.

TABLE 5.4.1

Bronchiolitis assessment tool (BAT)

	Mild	*Moderate*	*Severe*
Wheeze	None or end expiratory	Entire expiration	Inspiratory and expiratory
Feeding	Normal	Less than usual	Not interested, gasping/coughing
Oxygen	No oxygen requirement	May require oxygen	Requires oxygen
Chest indrawing	Nil/mild	Intercostal and/or tracheosternal	Severe with nasal flaring
Behavior	Normal	Irritability/lethargy	Irritability/lethargy

Flow chart 5.4.1 Management of bronchiolitis


Mild
Moderate
Severe
Alert pink in room air, saturation > 93% nil/mild respiratory signs, well hydrated. Feeding without shortness of breath
Any one or more of: Poor feeding Lethargy Marked respiratory signs Saturation <94%
As per moderate plus: O_2 requirement > 35% severe respiratory signs signs of tiring/CO_2 retention–sweaty, irritable apnea with cyanosis
Nasal saline nose drops before feeds observed oral feeds
Nasal saline nose drops consider NG supplemental feeds O_2 via nasal prong/mask suctioning PRN fluid balance consider chest X-ray
Consider chest X-ray, CBC blood culture. NPA, ABG if facility available nil by mouth, IV/NG feeds O_2 via mask/head box fluid balance, minimal handling
Normal feeds No risk factor
Risk factor present
Discharge home, follow up in next 48 hours
Admit for observation and O_2, NG feeds
Consider ICU admission for CPAP/ventilatory support

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5.5 ACUTE SEVERE ASTHMA

Jaydeep Choudhury, Jayanta Bandyopadhyay

A child with acute severe asthma may have the following features:

1. Cannot complete sentences in one breath or too breathless to talk or feed.
2. Pulse rate >130 in 2 to 5 years or >120 beyond 5 years.
3. Respiratory rate >50/min in 2 to 5 years or >30 beyond 5 years.
4. Peak expiratory flow (PEF) 33 to 50 percent of self best or predicted value.

Management

The first move should be to relieve the bronchospasm by nebulization or inhaler therapy.

1. Oxygen by mask at 5 litre/min to achieve SpO_2 >90 percent.
2. Metered dose inhaler (Fig. 5.5.1)—This is a very effective device to control acute exacerbation of asthma. Inhaler therapy may be started at home even before bringing the child to the hospital. If the child is a known asthmatic, the parents and the child should be educated about its use in emergency situations and they should always carry an inhaler with them. Salbutamol 100 mg per dose or levosalbutamol 50 mg per dose. The recommended schedule is 4–10 puffs at 20 minutes intervals, 3 times, that is over the first hour.

   With proper usage like using spacer device in older children or spacer with mask in small child most of the cases may be controlled effectively.
3. Nebulization: Salbutamol 0.15 mg/kg/dose with 2 to 3 mL normal saline.
   2.5 mg (0.5 mL) in children <20 kg body weight
   5 mg (1 mL) in children >20 kg body weight
   May be repeated every 20 minutes for 3 doses
   Continuous salbutamol nebulization may be applied 10 mg in 10 mL normal saline via a jet nebulizer.

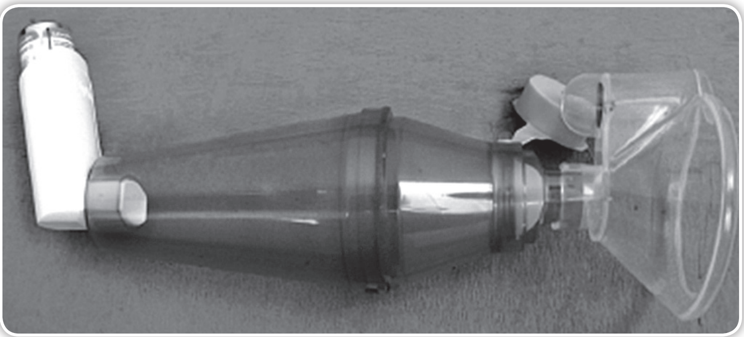


Figure 5.5.1 Metered dose inhaler with spacer and baby mask

4. Corticosteroid: A single oral corticosteroid, prednisolone 1 to 2 mg/kg may be given early. In older children as the body weight is more even 0.5 mg/kg dose is sufficient.

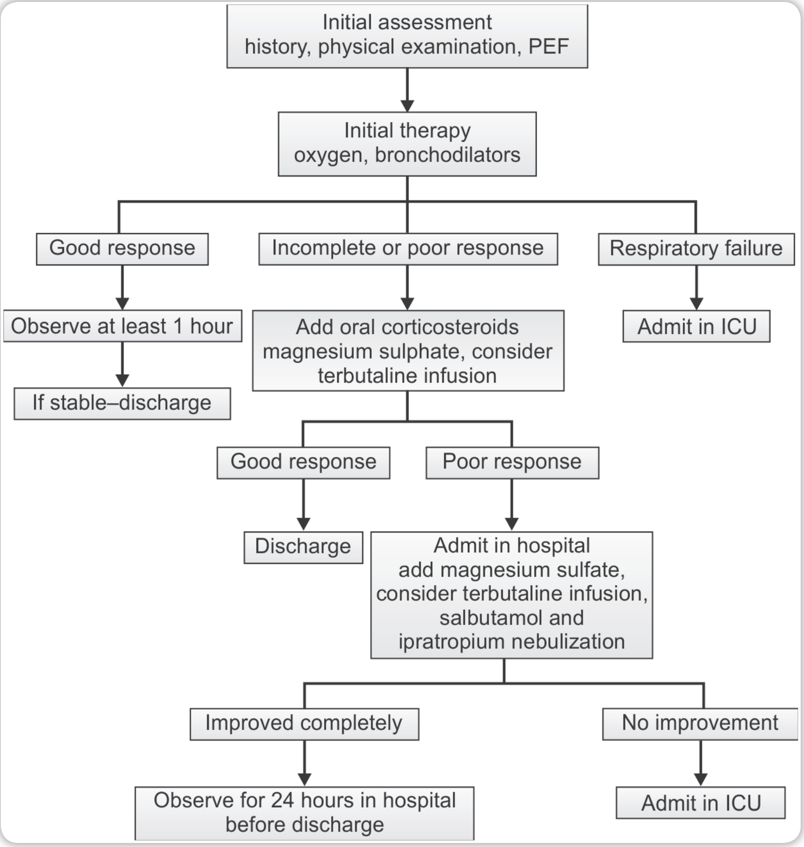
   The management algorithm of acute asthma is shown in Flow chart 5.5.1.

Good Response Group

If the child improves with the above measures and SpO_2 >90 percent in room air the child may be discharged and advised to take inhaled bronchodilators and oral corticosteroid like prednisolone 1 to 2 mg/kg/day in 2 divided doses for 3 days.

If the child does not respond to the above measure, a thorough evaluation and need to transfer to intensive care set up has to be considered.

Flow chart 5.5.1 Management of severe asthma



Signs of High Risk

1. Altered sensorium (either drowsy or irritable)
2. Bradycardia
3. Poor pulse volume
4. Cyanosis (with 60% oxygen)
5. Exhaustion (vocalization limited to 1–2 words) and excessive use of accessory muscles of respiration
6. Excessive diaphoresis
7. Silent chest on auscultation
8. ABG: $pCO_2 > 55$ mm Hg, $pO_2 < 60$ mm Hg, rate of rise of $pCO_2 > 5$ mm Hg/h, metabolic acidosis (base deficit > 7–10).

Poor Response Group

1. Continue oxygen.
2. Nebulization—salbutamol every 20 minutes.
3. Intravenous fluid.
4. Corticosteroid: If the child tolerates oral medicines, then steroid may be given orally. Parenterally methylprednisolone 2 mg/kg followed by 1 mg/kg every 6 hourly or hydrocortisone 10 mg/kg followed by 5 mg/kg 6 hourly intravenous.

   It is ideal to use corticosteroid early as it limits morbidity. With clinical improvement intravenous corticosteroid may be tapered after 48 hours. Inhaled corticosteroids are of not much benefit in acute asthma.
5. Ipratropium bromide: When used along with salbutamol it offers additional bronchodilator benefit. Dose is 0.5 mL in <1 year and 1 mL in >1 year nebulization along with normal saline.
6. β_2 agonist: Terbutaline or salbutamol may be used. Terbutaline 5 to 10 mg/kg intravenous bolus over 10 minutes followed by 2 to 10 mg/kg/h. The infusion should be under continuous ECG monitor. In case of tachycardia, arrhythmia, ST changes the infusion should be reduced and nebulization stopped.
7. Aminophylline: 5 to 6 mg/kg loading dose slow intravenous infusion followed by maintenance dose 0.5 to 1 mg/kg/h. Dose of terbutaline should be halved if aminophylline is concurrently administered.
8. Magnesium sulfate 50 percent solution: 25 to 50 mg/kg diluted with normal saline and infused over 30 minutes.

Same management should be continued for at least 4 hours. Hourly clinical assessment has to be done to detect any improvement or deterioration.

Improvement at the End of 6 Hours

1. Decrease β_2 agonist every 1 to 4 hourly.
2. Stop injection aminophylline.
3. Continue corticosteroid for 3 days.

Condition Unchanged

Consider mechanical ventilation.

Clinical Deterioration

Indications for mechanical ventilation:
1. Exhaustion, shallow respiration and drowsiness at presentation
2. Not responsive to above treatment
3. Severe respiratory distress, silent chest
4. Cardiorespiratory arrest
5. Comatosed child
6. ABG—PCO_2 >55 mm Hg, PO_2 <60 mm Hg, rate of rise of PCO_2 >5 mm Hg/h, metabolic acidosis (base deficit >7 – 10).

First Attack of Status Asthmaticus

The diagnoses to be considered are the following:
1. Foreign body
2. Massive aspiration
3. Pulmonary edema
4. CNS disorder (central hyperventilation)
5. Metabolic acidosis (DKA, inborn error of metabolism).

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6

Cardiovascular Emergencies

Mahasweta Chaudhuri, Jaydeep Choudhury, Jayanta Bandyopadhyay, Joshi Anand Karketta, Amitabha Chattopadhyay, Biswajit Bandopadhyay

6.1 CYANOTIC SPELL

Mahasweta Chaudhuri, Jaydeep Choudhury, Jayanta Bandyopadhyay

Cyanotic spell occurs most frequently in young infants with tetralogy of Fallot but may be associated with other congenital heart defects that have decreased pulmonary blood flow or a manifestation of shunt malfunction in whom palliative procedure (e.g. BT shunt, which is a systemic-pulmonary connection) has been done. Cyanotic spell can occur at any age.

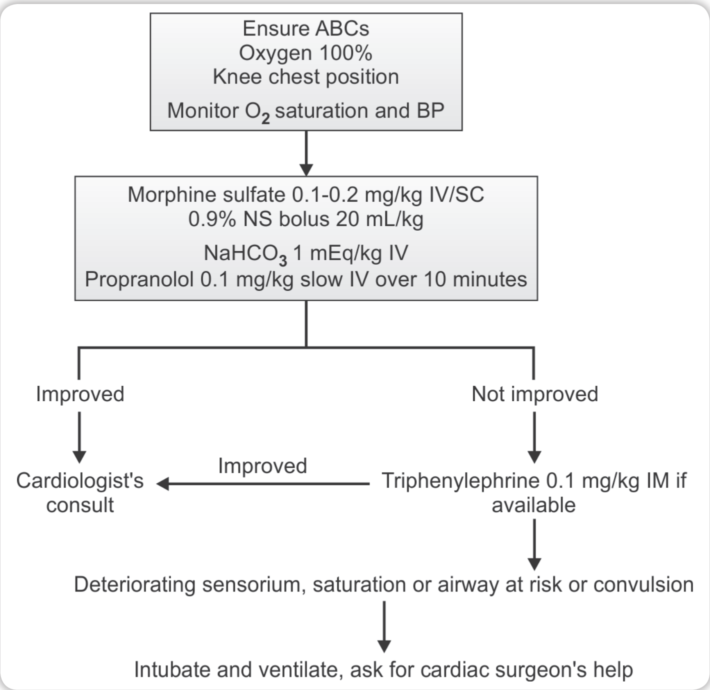
Management

Acute Care

Treatment of spells involves the following procedure. The algorithmic approach to management of cyanotic spell is shown in Flow chart 6.1.1.

1. ***Knee chest position:*** To increase systemic vascular resistance. This can be easily done in older children. Infants and small children can be picked up in the lap with legs folded at the knees to obtain similar result.
2. ***Oxygen:*** To be given at highest possible concentration though result may not be proportionate.
3. ***Morphine:*** 0.1 to 0.2 mg/kg subcutaneously. Also can be given IV 0.05 to 0.1 mg/kg slowly over 10 minutes. Morphine depresses respiratory center which in turn decreases systemic venous return. While giving morphine, facilities for ventilation should be available.
4. ***IV fluids:*** Preferably initially as bolus of 10 to 20 cc/kg which may be increased to 60 cc/kg. Bolus fluid should be isotonic saline or colloid. Extra

Flow chart 6.1.1 Algorithm for management of cyanotic spell


Ensure ABCs
Oxygen 100%
Knee chest position
Monitor O_2 saturation and BP
Morphine sulfate 0.1-0.2 mg/kg IV/SC
0.9% NS bolus 20 mL/kg
$NaHCO_3$ 1 mEq/kg IV
Propranolol 0.1 mg/kg slow IV over 10 minutes
Improved
Not improved
Cardiologist's consult
Improved
Triphenylephrine 0.1 mg/kg IM if available
Deteriorating sensorium, saturation or airway at risk or convulsion
Intubate and ventilate, ask for cardiac surgeon's help

volume can be given in cyanotic spell as the physiology is not inductive to CCF and also because of a restrictive RV physiology.

5. ***Sodium bicarbonate injection:*** 1 to 2 µg/kg slowly intravenous. This is to correct metabolic acidosis.
6. ***Propanolol:*** 0.1 to 0.2 mg/kg intravenously over 5 minutes. Propanolol reduces dynamic RV outflow obstruction, slows heart rate which in turn decreases right to left shunting. It also causes slight increase in systemic vascular resistance and blocks hyperpnea response.
7. ***Metoprolol injection:*** 0.1 mg/kg over 5 minutes, repeat every 5 minutes to max 3 doses , then start infusion 1 to 5 µg/kg/min. Metoprolol is only used when intravenous propranolol is not available.
8. Another short acting beta-blocker that can be used is esmolol injection 0.5 mg/kg over 1 minutes then 50 µg/kg/min over 4 minutes.
9. Transfuse packed red blood cell—5 to 10 mL/kg IV over 5 hours.
10. Treat sepsis if there is any evidence.
11. Mechanical ventilation if refractory to above treatment and perpetuating to bradycardia and/or spiralling desaturation, coma, convulsion. If ultimately ventilated, ketamine has a good role to play. Ketamine 0.25 to 1.0 mg/kg IV, has dual benefit. It causes sedation and increases systemic vascular resistance. Ketamine without ventilation may be tried but all arrangement to intubate and ventilate must be ready.
12. Consult a cardiac surgeon for emergency surgical intervention (BT shunt).

Ongoing Care

If there is a single severe or frequent minor spells, commence oral propranolol 0.5 mg/kg/dose TDS. This should be done in hospital, watching for hypotension and hypoglycemia. Increase gradually to 1.0 to 1.5 mg/kg/dose TDS (3.0–4.5 mg/kg/day).

Bibliography

1. American Heart Association Guidelines 2001.
2. Moss, Adams. Heart Disease in Infants, Children and Adolescents, 6th edition.
3. Park MK. Pediatric Cardiology for Practitioners, 3rd edition.
4. The Harriet Lane Handbook, 16th edition.

6.2 HEART FAILURE

Joshi Anand Karketta, Jayanta Bandyopadhyay, Jaydeep Choudhury

General Management

1. ABC, oxygen inhalation
2. Connect to a cardiac monitor
3. Secure an IV line
4. If in shock intubate and ventilate
5. Keep fluid input/output chart
6. Fluid restriction 70 percent mL/kg/day
7. If the baby is tachypneic consider feeding via NG tube.
8. Monitor serum electrolytes frequently (especially potassium).

Stepwise management of heart failure is shown in Flow chart 6.2.1 and the management of heart failure with shunt lesions is shown in Flow chart 6.2.2.

Specific Management

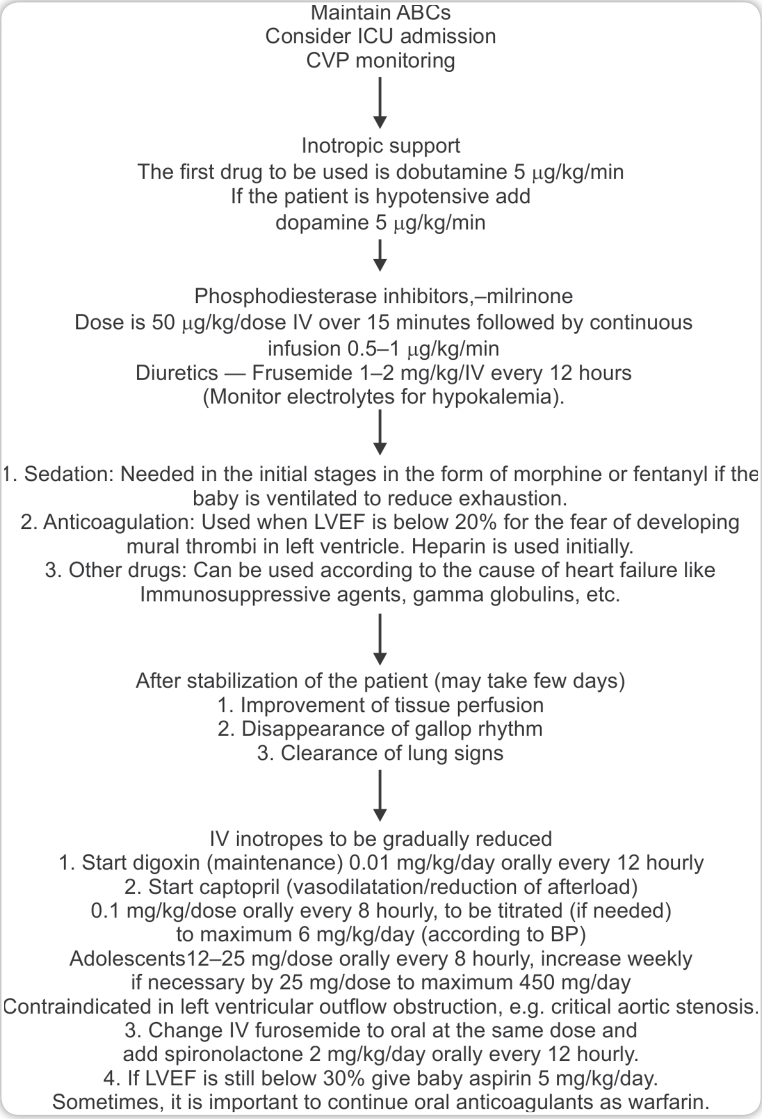
1. Dopamine is not used as first line inotropes unless the baby is hypotensive, it causes tachycardia and subsequently reduced tissue perfusion.
2. Milrinone—produces inotropic, vasodilator effect and afterload reduction. Best used as infusion in combination with dobutamine.
3. Monitor electrolytes for hypokalemia during frusemide administration.
4. Captopril can be increased weekly if necessary by 25 mg/dose to maximum 450 mg/day. Contraindicated in left ventricular outflow obstruction, e.g. critical aortic stenosis.

If captopril is given with digoxin and diuretics, then spironolactone should be reduced or stopped according to potassium level.

Important Considerations

1. When starting digoxin with diuretics (frusemide and spironolactone), the dose given as 0.01 mg/kg/day every 12 hourly.
2. If captopril is given with digoxin and diuretics, then spironolactone should be reduced or stopped according to potassium level.
3. Digoxin toxicity can occur if the above 4 drugs are given and lower doses of digoxin should be given (0.0075 mg/kg/day every 12 hourly).
4. It is advisable that antifailure therapy be started by cardiologist initially in conditions of large left to right shunt lesions and in obstructive lesions.

Flow chart 6.2.1 Management of heart failure due to acute myocarditis



Maintain ABCs
Consider ICU admission
CVP monitoring

↓

Inotropic support
The first drug to be used is dobutamine 5 μg/kg/min
If the patient is hypotensive add
dopamine 5 μg/kg/min

↓

Phosphodiesterase inhibitors,–milrinone
Dose is 50 μg/kg/dose IV over 15 minutes followed by continuous infusion 0.5–1 μg/kg/min
Diuretics — Frusemide 1–2 mg/kg/IV every 12 hours
(Monitor electrolytes for hypokalemia).

↓

1. Sedation: Needed in the initial stages in the form of morphine or fentanyl if the baby is ventilated to reduce exhaustion.
2. Anticoagulation: Used when LVEF is below 20% for the fear of developing mural thrombi in left ventricle. Heparin is used initially.
3. Other drugs: Can be used according to the cause of heart failure like Immunosuppressive agents, gamma globulins, etc.

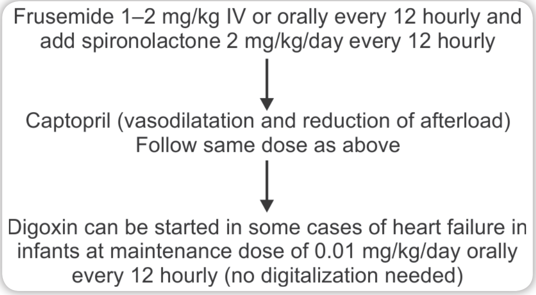
↓

After stabilization of the patient (may take few days)
1. Improvement of tissue perfusion
2. Disappearance of gallop rhythm
3. Clearance of lung signs

↓

IV inotropes to be gradually reduced
1. Start digoxin (maintenance) 0.01 mg/kg/day orally every 12 hourly
2. Start captopril (vasodilatation/reduction of afterload)
0.1 mg/kg/dose orally every 8 hourly, to be titrated (if needed) to maximum 6 mg/kg/day (according to BP)
Adolescents12–25 mg/dose orally every 8 hourly, increase weekly if necessary by 25 mg/dose to maximum 450 mg/day
Contraindicated in left ventricular outflow obstruction, e.g. critical aortic stenosis.
3. Change IV furosemide to oral at the same dose and add spironolactone 2 mg/kg/day orally every 12 hourly.
4. If LVEF is still below 30% give baby aspirin 5 mg/kg/day.
Sometimes, it is important to continue oral anticoagulants as warfarin.

Flow chart 6.2.2 Management of heart failure due to shunt lesions (VSD, PDA, AVSD)



Frusemide 1–2 mg/kg IV or orally every 12 hourly and add spironolactone 2 mg/kg/day every 12 hourly

↓

Captopril (vasodilatation and reduction of afterload)
Follow same dose as above

↓

Digoxin can be started in some cases of heart failure in infants at maintenance dose of 0.01 mg/kg/day orally every 12 hourly (no digitalization needed)

Management of chronic heart failure (with dilated cardiomyopathy and left ventricular dysfunction)

1. Stable patients should be maintained on ACE inhibitors (e.g. Captopril, enalapril) on for long-term. The doses are adjusted according to BP.
2. Diuretics are given in some patients as adjunctive therapy when left ventricular ejection fraction is <40 percent.
3. Long acting beta blockers have proven efficacy in patient with chronic heart failure. The drug used nowadays is carvedalol 0.1 mg/kg/dose every12 hourly. Increase slowly and monthly by 0.1 mg/kg/dose to maximum dose of 6.25 mg every12 hourly.

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3. The Harriet Lane Handbook, 2002.
4. Treatment of heart failure in children. Current Paediatrics, 2005;15,539–48.

6.3 ABNORMAL PULSE RATE OR RHYTHM

Amitabha Chattopadhyay, Biswajit Bandopadhyay

Most children with cardiac arrhythmias present with vague and nonspecific symptoms. "Feeding difficulty" or "fussiness" is the common presenting features in infants and young children. They even present in shock. Cardiac arrhythmias may lead to cardiopulmonary compromise and arrest if left untreated.

The most common pediatric cardiac arrhythmias are sinus tachycardia (50%), supraventricular tachycardia (13%), bradycardia (6%) and atrial fibrillation (4.6%). The normal respiratory rate and heart rate in children are shown in Table 6.3.1.

General Management of Arrhythmia

Whatever may be the presenting feature, the basic approach remains almost the same. As with any other emergencies in children, the management starts with the primary assessment following the algorithm of ABCDE.

Airway

Maintain an open airway, may require intubation.

Breathing

Assess the effort of breathing, the efficacy of breathing, and the effects of respiratory failure on other systems.

TABLE 6.3.1

Normal respiratory and heart rate in children of different age groups

Age	*RR / min*	*HR/ min*
Birth	40–60	100–160
3 months	30–50	100–160
6 months	30–50	100–160
1 year	30–40	100–160
2 years	20–30	100–150
4 years	20	80–130
6 years	16	70–120
8 years	16	70–110
10 years	16	60–100
12 years	16	60–100
14 years	16	60–100

Features Suggesting Cardiac Cause of Respiratory Inadequacy

i. Cyanosis, not correcting with oxygen therapy
ii. Tachycardia out of proportion to respiratory difficulty
iii. Raised jugular venous pressure
iv. Gallop rhythm or murmur
v. Enlarged liver
vi. Inequality of pulses.

Circulation

Assessment of circulatory status is very important. An abnormal pulse rate is defined as one falling outside the normal range.

Important Considerations

i. If there is shock and the heart rate <60/min, start chest compressions.
ii. ECG shows ventricular tachycardia then give up to 1 to 2 synchronous electrical shocks at 1and 2 joules/kg. Use sedation or anesthesia for the child who is responsive to pain. Synchronous shocks for VT may be ineffectual if the defibrillator cannot recognize the abnormally shaped QRS complex. In such a situation, one may have to deliver asynchronous shock, because without conversion the rhythm may deteriorate to VF and asystole. Synchronization avoids shock delivery at a point in the cardiac cycle likely to precipitate ventricular fibrillation.
iii. Gain intravenous or intraosseous access.
iv. If the tachyarrhythmia is SVT then give intravenous/intraosseous adenosine 100 μg/kg to a maximum single dose of 500 μg/kg (300 μg/kg under one month) if this can be administered more quickly than a synchronous electrical shock.
v. Send blood for CBC, urea, creatinine, glucose and other laboratory tests.
vi. Give a bolus of 20 mL/kg IV of crystalloid to a patient with bradycardia who is in shock.

Bradycardia

Bradycardia in children is almost always a preterminal finding in patients with respiratory or circulatory insufficiency. Airway, breathing and circulation should always be assessed before any other forms of treatment.

Treatment—if there is hypoxia and shock:

1. High concentration of oxygen, bag mask ventilation, intubation and IPPV.
2. Volume expansion (20 mL/kg of 0.9% saline repeated as recommended in the treatment of shock).
3. If ineffective give bolus of adrenaline 10 μg/kg IV.
4. If ineffective give infusion of adrenaline 0.05 to 2 μg/kg/min IV.

Adrenaline induces vasoconstriction increasing aortic diastolic pressure and thus the coronary perfusion pressure, a critical determinant of successful resuscitation.

Treatment—if there has been vagal stimulation:

1. Adequate ventilation.
2. Atropine 20 μg/kg IV/IO (minimum dose 100 micrograms maximum dose 600 micrograms). It is actually a parasympatholytic drug that accelerates sinus or atrial pacemakers and increases AV conduction. Small doses of atropine (0.1 mg) may produce paradoxical bradycardia, hence the minimum dose is required.
3. It may be repeated in 5 minutes (maximum total dose of 1.0 mg in a child and 2.0 mg in an adolescent).
4. If IV/IO access is unavailable, atropine (0.04 mg/kg) may be administered intratracheally (unreliable absorption).
5. If there has been poisoning, seek expert toxicology help.

If the rhythm does not return to a sinus rhythm after the initial resuscitative efforts and the patient remains hemodynamically unstable, one may have to transfer the patient to a specialist cardiac center where facilities of a temporary pacemaker insertion is available, pending further investigations regarding the exact cause of the bradycardia .

PALS bradycardia protocol is cited below in Flow chart 6.3.1 as a guide.

Tachycardia

If there are no palpable pulses, proceed with the PALS Pulseless Arrest Algorithm (Flow chart 6.3.1). If pulses are palpable and the patient has signs of hemodynamic compromise (poor perfusion, tachypnea, weak pulses), ensure that the ABC is maintained, administer supplementary oxygen, and attach an ECG monitor or defibrillator to assess the rhythm and QRS duration.

QRS Complexes

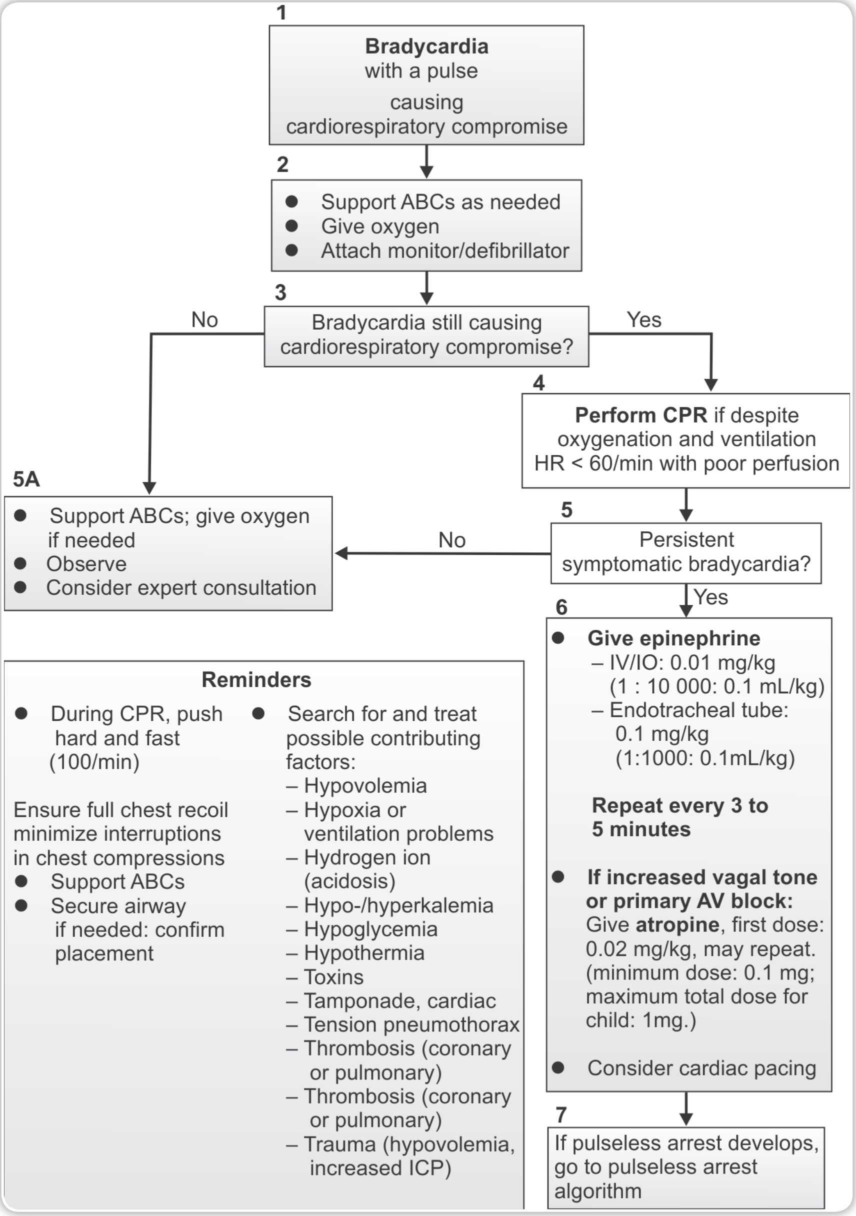
QRS duration is ≤0.08 second is narrow-complex tachycardia. Narrow complex tachycardias are universally of supraventricular origin. QRS duration ≥0.08 second is wide-complex tachycardia.

Narrow Complex Tachycardias

Evaluate a 12-lead ECG (best in lead II and V1) and the patient's clinical presentation and history to differentiate probable sinus tachycardia from probable supraventricular tachycardia (SVT). If the rhythm is sinus tachycardia, search for and treat reversible causes.

If the patient is very unstable or IV, access is not readily available, provide electrical synchronized cardioversion. Consider sedation if possible. Starting

Flow chart 6.3.1 PALS bradycardia protocol


1
Bradycardia with a pulse causing cardiorespiratory compromise
2
● Support ABCs as needed
● Give oxygen
● Attach monitor/defibrillator
3
Bradycardia still causing cardiorespiratory compromise?
No
Yes
4
Perform CPR if despite oxygenation and ventilation HR < 60/min with poor perfusion
5A
● Support ABCs; give oxygen if needed
● Observe
● Consider expert consultation
5
Persistent symptomatic bradycardia?
No
Yes
6
● Give epinephrine
– IV/IO: 0.01 mg/kg (1 : 10 000: 0.1 mL/kg)
– Endotracheal tube: 0.1 mg/kg (1:1000: 0.1mL/kg)
Repeat every 3 to 5 minutes
● If increased vagal tone or primary AV block: Give atropine, first dose: 0.02 mg/kg, may repeat. (minimum dose: 0.1 mg; maximum total dose for child: 1mg.)
● Consider cardiac pacing
7
If pulseless arrest develops, go to pulseless arrest algorithm
Reminders
● During CPR, push hard and fast (100/min)
Ensure full chest recoil minimize interruptions in chest compressions
● Support ABCs
● Secure airway if needed: confirm placement
● Search for and treat possible contributing factors:
– Hypovolemia
– Hypoxia or ventilation problems
– Hydrogen ion (acidosis)
– Hypo-/hyperkalemia
– Hypoglycemia
– Hypothermia
– Toxins
– Tamponade, cardiac
– Tension pneumothorax
– Thrombosis (coronary or pulmonary)
– Thrombosis (coronary or pulmonary)
– Trauma (hypovolemia, increased ICP)

dose of electrical cardioversion 0.5 to 1 J/kg. If unsuccessful, repeat using a dose of 2 J/kg.

If a second shock is unsuccessful or the tachycardia recurs quickly, consider antiarrhythmic therapy (amiodarone or procainamide) before a third shock. Consult a pediatric cardiologist.

Consider amiodarone or procainamide for SVT unresponsive to vagal maneuvers and adenosine.

If the presentation is of a pulseless condition with shock, the preferable guideline is the PALS pulseless arrest protocol, as cited in Flow chart 6.3.2.

Supraventricular Tachycardia

Supraventricular tachycardia (SVT) is the most common non-arrest arrhythmia during childhood and is the most common arrhythmia that produces cardiovascular instability during infancy. SVT in infants generally produces a heart rate >220/min. Figure 6.3.1. shows an ECG of supraventricular tachycardia.

Management

Reassess ABC.

Vagal Stimulation

It should be done with ECG monitoring. The following are the maneuvers to increase the vagal tone which slows the atrioventricular conduction to interrupt the tachycardia.

i. Application of ice or iced water over the face to elicit the "Diving reflex"
ii. One-sided carotid body massage
iii. Older children can try a Valsalva maneuver. Some children may become experienced to know that a certain position or action will usually cause a return to sinus rhythm. Blowing hard through a partially obstructed straw may be effective for some children.

Ocular pressure in an infant or child is contraindicated as retinal damage may result.

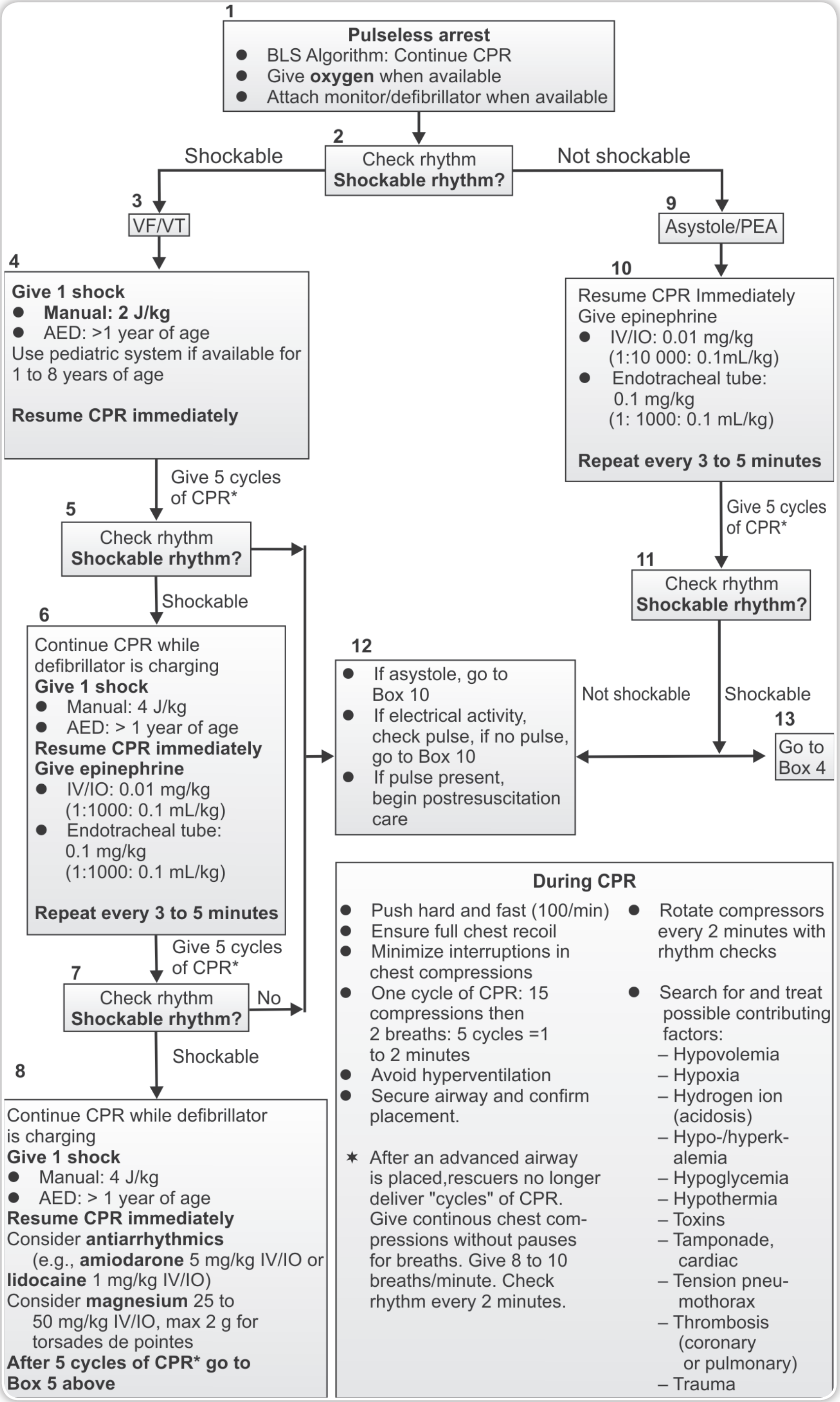
Adenosine

If the tachycardia does not terminate with these maneuvers then intravenous adenosine should be given. Adenosine is the drug of choice for supraventricular tachycardia. Adenosine is a very rapidly acting drug with a half-life of less than 10 seconds.

Dose: 100 μg/kg rapid intravenously. If there is no response, 200 μg/kg and then 300 μg/kg may be given. The effect is short lasting and the tachycardia might recur. It should be rapidly injected into a large peripheral vein, using a 3 way stopcock followed by a rapid saline flush before it is taken up by the RBCs for metabolism.

The maximum total dose is 500 μg/kg (300 μg/kg under 1 month) up to a maximum of 12 mg in total.

Flow chart 6.3.2 PALS pulseless arrest protocol



1 **Pulseless arrest**
- BLS Algorithm: Continue CPR
- Give **oxygen** when available
- Attach monitor/defibrillator when available

2 Check rhythm
Shockable rhythm?

3 VF/VT

4 **Give 1 shock**
- **Manual: 2 J/kg**
- AED: >1 year of age

Use pediatric system if available for 1 to 8 years of age

Resume CPR immediately

Give 5 cycles of CPR*

5 Check rhythm
Shockable rhythm?

6 Continue CPR while defibrillator is charging
Give 1 shock
- Manual: 4 J/kg
- AED: > 1 year of age

Resume CPR immediately
Give epinephrine
- IV/IO: 0.01 mg/kg (1:1000: 0.1 mL/kg)
- Endotracheal tube: 0.1 mg/kg (1:1000: 0.1 mL/kg)

Repeat every 3 to 5 minutes

Give 5 cycles of CPR*

7 Check rhythm
Shockable rhythm?

8 Continue CPR while defibrillator is charging
Give 1 shock
- Manual: 4 J/kg
- AED: > 1 year of age

Resume CPR immediately
Consider **antiarrhythmics** (e.g., **amiodarone** 5 mg/kg IV/IO or **lidocaine** 1 mg/kg IV/IO)
Consider **magnesium** 25 to 50 mg/kg IV/IO, max 2 g for torsades de pointes
After 5 cycles of CPR* go to Box 5 above

9 Asystole/PEA

10 Resume CPR Immediately
Give epinephrine
- IV/IO: 0.01 mg/kg (1:10 000: 0.1mL/kg)
- Endotracheal tube: 0.1 mg/kg (1: 1000: 0.1 mL/kg)

Repeat every 3 to 5 minutes

Give 5 cycles of CPR*

11 Check rhythm
Shockable rhythm?

12
- If asystole, go to Box 10
- If electrical activity, check pulse, if no pulse, go to Box 10
- If pulse present, begin postresuscitation care

13 Go to Box 4

During CPR
- Push hard and fast (100/min)
- Ensure full chest recoil
- Minimize interruptions in chest compressions
- One cycle of CPR: 15 compressions then 2 breaths: 5 cycles =1 to 2 minutes
- Avoid hyperventilation
- Secure airway and confirm placement.

✶ After an advanced airway is placed,rescuers no longer deliver "cycles" of CPR. Give continous chest compressions without pauses for breaths. Give 8 to 10 breaths/minute. Check rhythm every 2 minutes.

- Rotate compressors every 2 minutes with rhythm checks
- Search for and treat possible contributing factors:
  - Hypovolemia
  - Hypoxia
  - Hydrogen ion (acidosis)
  - Hypo-/hyperkalemia
  - Hypoglycemia
  - Hypothermia
  - Toxins
  - Tamponade, cardiac
  - Tension pneumothorax
  - Thrombosis (coronary or pulmonary)
  - Trauma

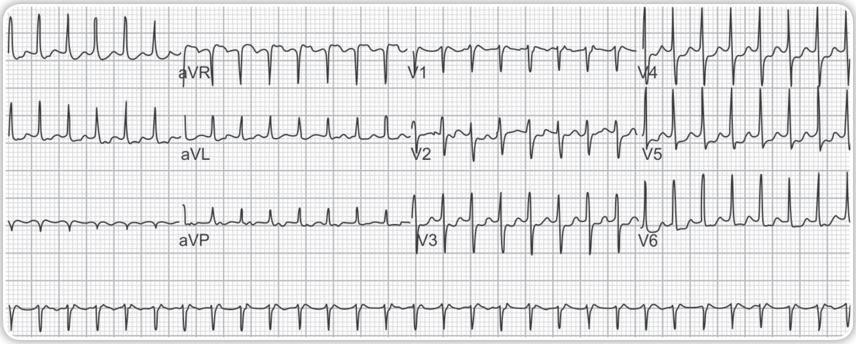


Figure 6.3.1 ECG showing supraventricular tachycardia

TABLE 6.3.2

Characteristics to distinguish between sinus tachycardia and SVT

Sinus tachycardia	*Supraventricular tachycardia*
Infants and children usually have a HR <220/min	Usually HR >220/min
P waves if visible usually upright in leads I and AVF	P waves if visible are usually negative in leads II, III and AVF in SVT
Beat-to-beat variability of HR present, which is often responsive to stimulation	No beat-to-beat variability of HR in SVT
HR slows gradually in response to treatment	Termination is abrupt
Clinical scenario of shock with preceding history of age, blood loss, septicemia, etc.	Clinical scenario usually points towards some pre-existent or co-existent cardiac disorders

Cardioversion

The patient can be treated by cardioversion with a general anesthesia or sedation or a rapid atrial overdrive pacing, if pacing wires are in place in a postoperative patient or by help of transcutaneous pacing pads.

To start with a dose of 0.5 to 1 J/kg may be used and repeated using a dose of 2 J/kg.

Further Management

If the SVT does not terminate by these methods, one of the following may be suggested but it is preferable to consult a pediatric cardiologist before doing so.

1. *Amiodarone:* Loading dose of 5 mg/kg over at least 1 to 2 hours, followed by a continuous infusion of 5 to15 microgram/kg/min.
2. *Procainamide and flecainide:* These can be used but preferably at a specialized center.

3. *Propranolol:* Dose of 50 μg slowly intravenously. This may be tried only if pacing is available, as asystole may occur. Propranolol and verapamil should not be given to the same patient. It can be combined with digoxin.
4. *Verapamil:* This drug has been associated with irreversible hypotension and asystole when given to infants. It therefore should not be used in children under first year of age and with caution in children as may cause hypotension and myocardial depression.

Wide-Complex Tachycardia

Wide-complex tachycardia with poor perfusion is probably ventricular in origin but may be supraventricular with aberrancy. All wide-complex tachycardias should be considered as ventricular tachycardias unless proved otherwise. ECG of ventricular tachycardia is shown in Figure 6.3.2.

Approach to a Child with VT

Emergency Treatment of VT

1. Reassess ABC.
2. Hemodynamically stable child with ventricular tachycardia—early consultation with a pediatric cardiologist is preferred.
3. Amiodarone (5 mg/kg over minutes to over an hour depending on the urgency) or intravenous procainamide (15 mg/kg over 30–60 minutes, monitor ECG and BP). Amiodarone slows AV conduction, prolongs the AV refractory period and QT interval, and slows ventricular conduction

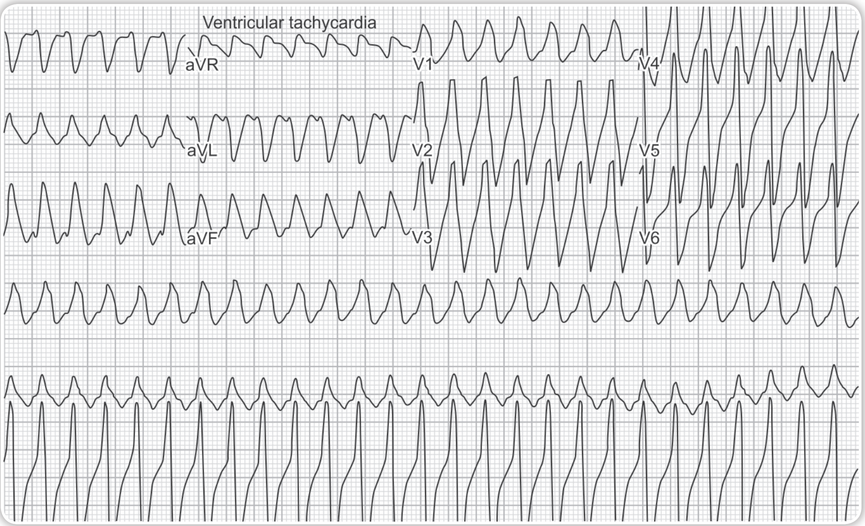


Figure 6.3.2 ECG showing ventricular tachycardia

(thus widening the QRS). Complications include bradycardia, heart block, and torsades de pointes ventricular tachycardia (VT). Be careful when administering with another drug causing QT prolongation. Procainamide works by prolonging the refractory period of the atria and ventricles and depressing the conduction velocity. Both of them can cause hypotension, through vasodilatation, which should be treated with volume expansion.

4. In cases, where the ventricular arrhythmia has been caused by drug toxicity, DC shock after sedation/anesthesia may be the safest approach. Use synchronous shocks initially, which are less likely to produce ventricular fibrillation. If synchronous shocks are ineffectual (if the instrument fails to identify the abnormal QRS complexes), try asynchronous if the child is in shock.
5. For torsade de pointes ventricular tachycardia: Magnesium sulphate as IV infusion (over several minutes) of 25–50 mg/kg (up to 2 g). Magnesium produces vasodilatation and may cause hypotension if administered rapidly.
6. Amiodarone 5 mg/kg may be given over a few minutes in ventricular tachycardia if the child is in severe shock.
7. Lidocaine decreases automaticity and suppresses ventricular arrhythmias.

Hemodynamically Unstable

Treat with synchronized electrical cardioversion (0.5 to 1 J/kg). If it does not delay cardioversion, try a dose of adenosine. Then a second shock with 2 J/kg may be given.

If a second shock (2 J/kg) is unsuccessful or if the tachycardia recurs quickly, consider antiarrhythmic therapy (Amiodarone or procainamide) before a third shock.

It is important not to delay a safe therapeutic intervention for longer than necessary in VT as the rhythm often deteriorates quite quickly into pulseless VT or VF.

Shown in Flow chart 6.3.3 is the PALS tachycardia algorithm, which should be observed as a guide.

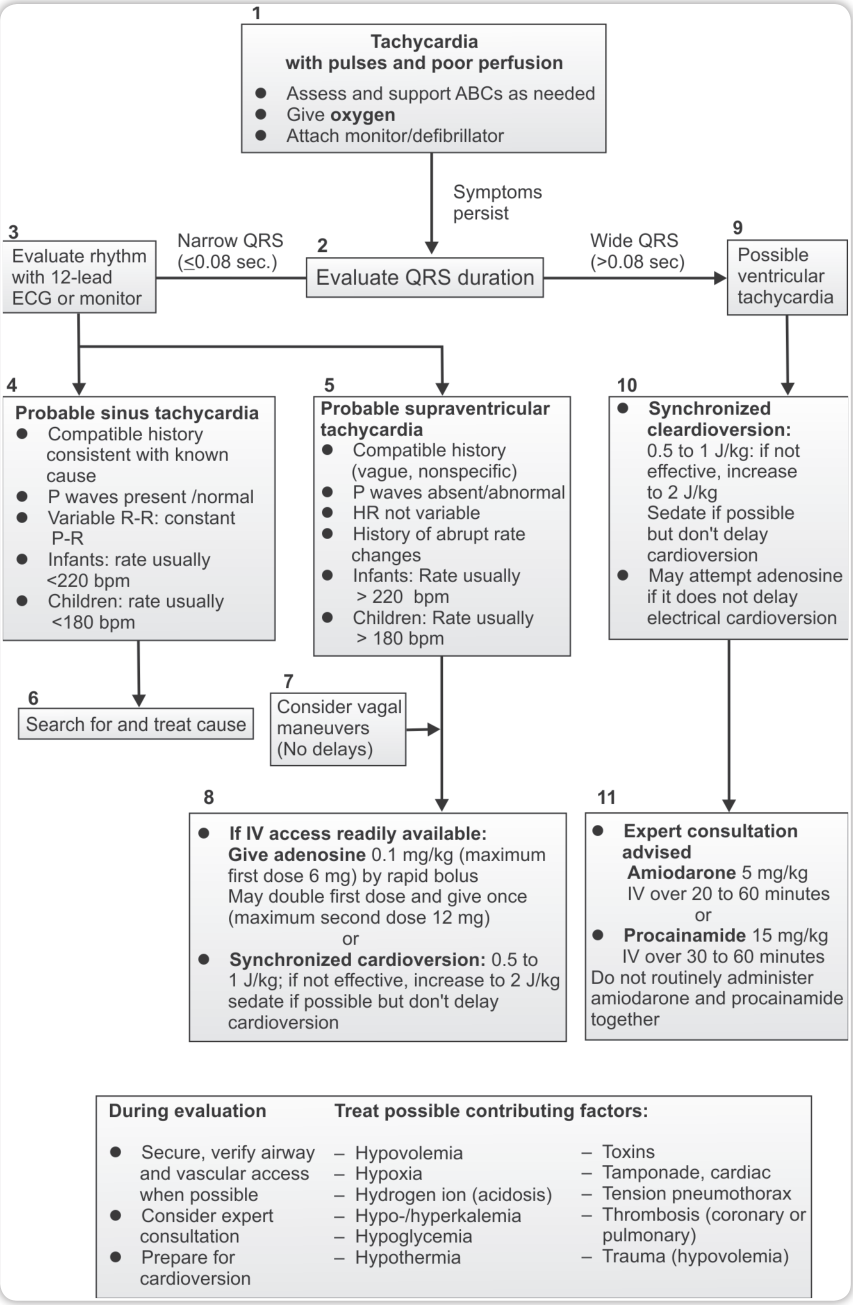
Defibrillator for Cardioversion

Defibrillators are either manual or automated (AED), with monophasic or biphasic waveforms. The energy adjustment of the machine should be appropriate for children.

Paddle Size

Use the largest paddles or self-adhering electrodes that will fit on the chest wall without touching (leave about 3 cm between the paddles). The best paddle size for adults and children weighing more than 10 kg is 8 to 10 cm. Infant paddles are for infants weighing less than10 kg.

Flow chart 6.3.3 PALS tachycardia algorithm


1
Tachycardia with pulses and poor perfusion
● Assess and support ABCs as needed
● Give oxygen
● Attach monitor/defibrillator
Symptoms persist
2
Evaluate QRS duration
Narrow QRS (≤0.08 sec.)
3
Evaluate rhythm with 12-lead ECG or monitor
Wide QRS (>0.08 sec)
9
Possible ventricular tachycardia
4
Probable sinus tachycardia
● Compatible history consistent with known cause
● P waves present /normal
● Variable R-R: constant P-R
● Infants: rate usually <220 bpm
● Children: rate usually <180 bpm
5
Probable supraventricular tachycardia
● Compatible history (vague, nonspecific)
● P waves absent/abnormal
● HR not variable
● History of abrupt rate changes
● Infants: Rate usually > 220 bpm
● Children: Rate usually > 180 bpm
10
● Synchronized cleardioversion: 0.5 to 1 J/kg: if not effective, increase to 2 J/kg Sedate if possible but don't delay cardioversion
● May attempt adenosine if it does not delay electrical cardioversion
6
Search for and treat cause
7
Consider vagal maneuvers (No delays)
8
● If IV access readily available: Give adenosine 0.1 mg/kg (maximum first dose 6 mg) by rapid bolus May double first dose and give once (maximum second dose 12 mg)
or
● Synchronized cardioversion: 0.5 to 1 J/kg; if not effective, increase to 2 J/kg sedate if possible but don't delay cardioversion
11
● Expert consultation advised Amiodarone 5 mg/kg IV over 20 to 60 minutes
or
● Procainamide 15 mg/kg IV over 30 to 60 minutes
Do not routinely administer amiodarone and procainamide together
During evaluation
● Secure, verify airway and vascular access when possible
● Consider expert consultation
● Prepare for cardioversion
Treat possible contributing factors:
– Hypovolemia
– Hypoxia
– Hydrogen ion (acidosis)
– Hypo-/hyperkalemia
– Hypoglycemia
– Hypothermia
– Toxins
– Tamponade, cardiac
– Tension pneumothorax
– Thrombosis (coronary or pulmonary)
– Trauma (hypovolemia)

Interface

The electrode-chest wall interface can be gel pads, electrode cream, paste, or self-adhesive monitoring-defibrillation pads. Saline-soaked pads, ultrasound gel, bare paddles, or alcohol pads should not be used.

Energy Dose

Energy doses 4 J/kg (up to 9 J/kg) have effectively defibrillated children.

Technique

1. Apply firm pressure on the paddles (manual) placed over the right side of the upper chest and the apex of the heart (to the left of the nipple over the left lower ribs). Alternatively place one electrode on the front of the chest just to the left of the sternum and the other over the upper back below the scapula.
2. Provide CPR until the defibrillator is ready to deliver a shock and resume CPR, beginning with chest compressions, immediately after shock delivery. Minimize interruptions of chest compressions.
3. Give 1 shock (2 J/kg) as quickly as possible and immediately resume CPR, beginning with chest compressions. Biphasic defibrillators have a first shock success rate that exceeds 90 percent.
4. CPR may provide some coronary perfusion with oxygen and substrate delivery, increasing the likelihood of defibrillation with a subsequent shock.
5. Continue CPR for about 5 cycles (about 2 minutes).

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7

Gastrointestinal Emergencies

Jaydeep Choudhury, Jayanta Bandyopadhyay

7.1 ACUTE ABDOMINAL PAIN

Jaydeep Choudhury

The first task in the management of a child with acute abdominal pain is to determine whether assessment by a surgeon is required. It is also worthwhile to remember that not only abdominal but also extra-abdominal emergencies produce acute abdomen. Children presenting with acute abdominal pain should be thoroughly evaluated before labelling them as a nonspecific or nonorganic disorder.

Important Considerations

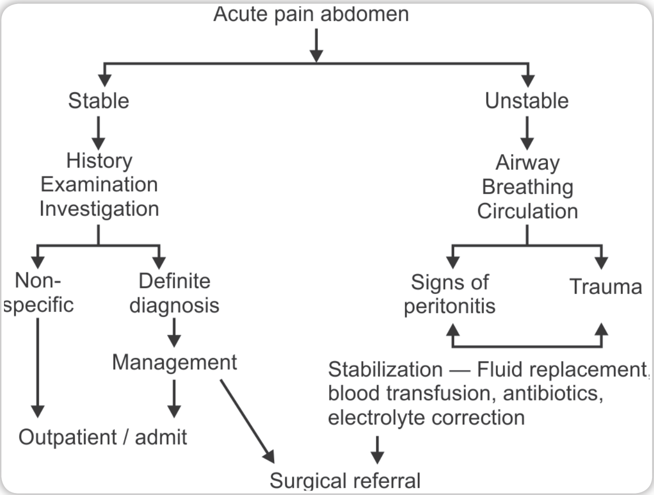
1. Surgical abdomen (abdominal distension, guarding, rigidity, absent peristalsis, lump)
2. Persistent vomiting or bilious vomiting
3. Dehydration or shock
4. It is often very difficult to distinguish between organic and nonorganic pain in children. The differentiating points are shown in Table 7.1.1. Approach to acute abdominal pain is shown in Flow chart 7.1.1.

TABLE 7.1.1

Differentiation of nonorganic from organic pain

	Organic	*Nonorganic*
Nature of pain	Any time in day and night	Periodic, usually in day, good in-between. Often periumbilical
History	1. Weight loss 2. Lack of energy 3. Fever 4. Change in bowel habit 5. Urinary symptoms 6. Intestinal symptoms 7. Vomiting: continuous, bile stained, hematemesis 8. Rectal bleeding	1. Migraine 2. School and family problem 3. Isolated vomiting, not bile stained
Examination	Various manifestations	Usually normal and thriving

Flow chart 7.1.1 Approach to acute abdominal pain



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7.2 UPPER GASTROINTESTINAL BLEEDING

Jaydeep Choudhury

Gastrointestinal (GI) bleeding manifests in the following ways:

1. Hematemesis is the passage of vomited material that is coffee grounds in color or contains frank blood.
2. Melena is the passage of black tarry stool, results from bacterial degradation of hemoglobin.
3. Hematochezia is passage of bright red color blood from the rectum. This can occur in lower as well as upper GI bleeding.

The age wise etiological agents are shown in Table 7.2.1 and the grades of GI bleeding are shown in Flow chart 7.2.1.

Management

Immediate

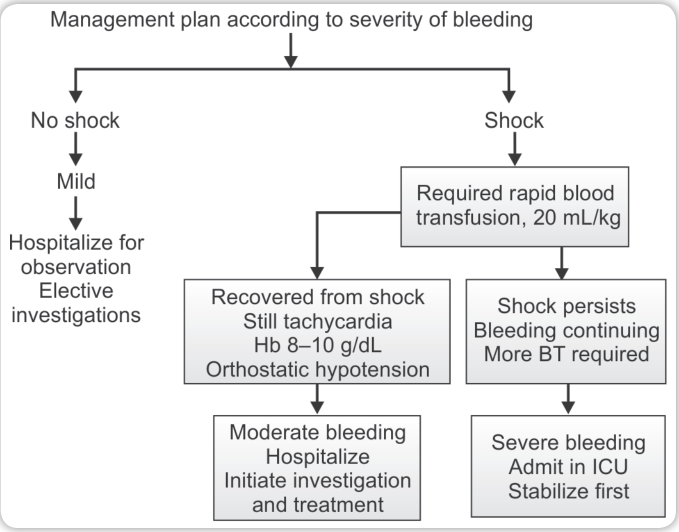
1. Double IV access
2. Oxygen inhalation
3. Nasogastric tube in situ
4. Blood drawn for investigations as detailed above
5. Urinary bladder catheterization to measure urine output
6. CVP line and keep pressure of 5 to 8 cm of water
7. Endotracheal intubation and ventilation if required.

TABLE 7.2.1

Etiology according to age

Neonates	*1 month to 1 year*	*1 year to 12 years*	*Adolescents*
1. Swallowed maternal blood	1. Gastritis	1. Esophageal varices	1. Esophageal varices
2. Hemorrhagic disease of newborn	2. Stress ulcer	2. Gastritis	2. Gastritis
3. Gastritis	3. Esophagitis	3. Stress ulcer	3. Stress ulcer
4. Stress ulcer	4. Mallory-weiss syndrome	4. Esophagitis	4. Esophagitis
5. Esophagitis	5. Vascular malformation	5. Mallory-weiss syndrome	5. Mallory-weiss syndrome
6. Congenital blood dyscrasias	6. GIT duplication	6. Peptic ulcer	6. Peptic ulcer
7. Vascular malformation			7. Foreign body
8. Idiopathic			

Flow chart 7.2.1 Management plan according to severity and bleeding



Restore and Maintain Intravascular Volume

1. Fluid bolus—10 to 20 mL/kg of normal saline or Ringer's lactate rapidly over 30 to 60 minutes
2. Repeat fluid boluses till urine output is normal and pulse, BP, CFT improves
3. Monitor and maintain hematocrit of 30 percent.

Re-establish a Normal Oxygen Carrying Capacity

Transfuse whole blood initially to maintain intravascular volume. Once bleeding stops and specific components are available, change to packed cell.

Determination of Source and Site of Bleeding

If possible determine source and site of bleeding and steps should be taken to stop bleeding. After initial stabilization and review of whole clinical situation, emergency endoscopy should be planned and approached as shown in Flow chart 7.2.2. The management plan of acute variceal bleed is shown in Flow chart 7.2.3.

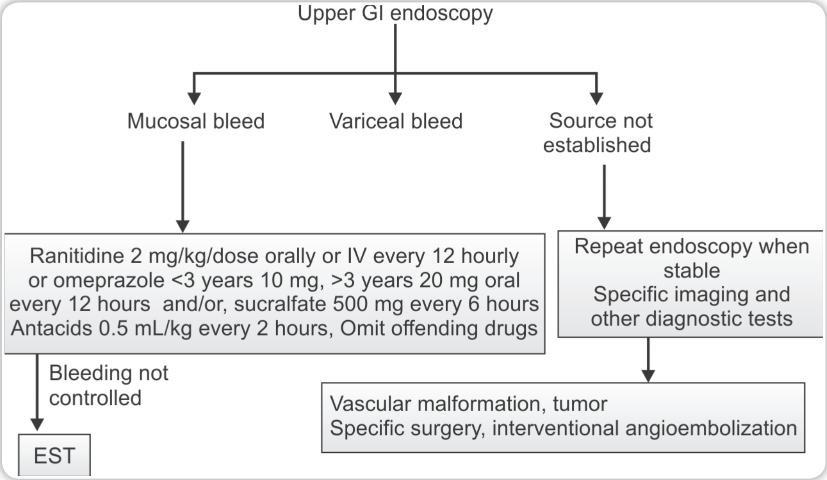
Abbreviations Used

EST – Endoscopic sclerotherapy

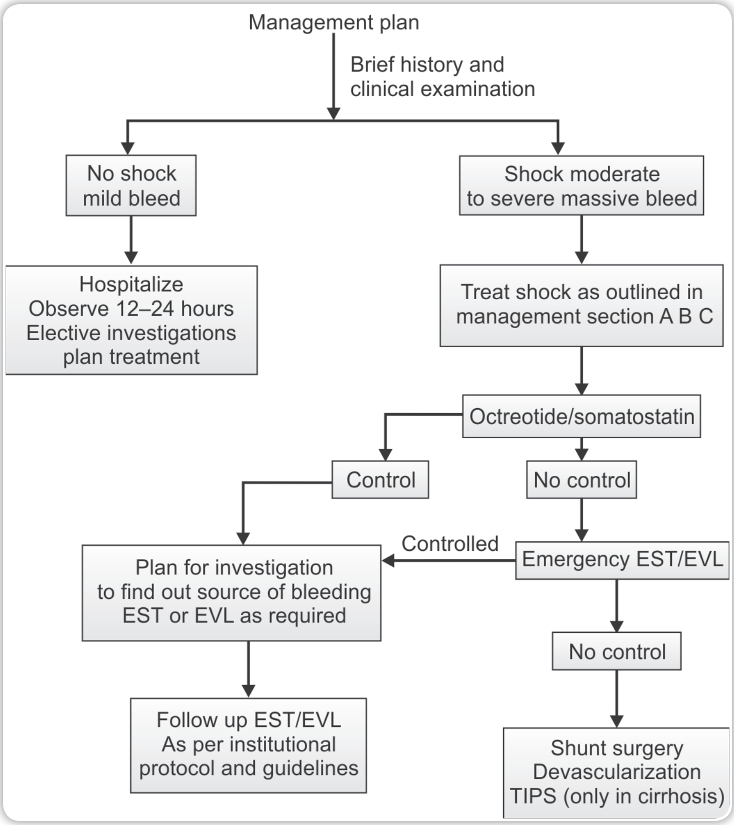
EVL – Endoscopic variceal ligation

TIPS – Transjugular intrahepatic portosystemic shunt.

Flow chart 7.2.2 Endoscopic evaluation of GI bleed


Upper GI endoscopy
Mucosal bleed
Variceal bleed
Source not established
Ranitidine 2 mg/kg/dose orally or IV every 12 hourly or omeprazole <3 years 10 mg, >3 years 20 mg oral every 12 hours and/or, sucralfate 500 mg every 6 hours Antacids 0.5 mL/kg every 2 hours, Omit offending drugs
Repeat endoscopy when stable
Specific imaging and other diagnostic tests
Bleeding not controlled
EST
Vascular malformation, tumor
Specific surgery, interventional angioembolization

Flow chart 7.2.3 Management plan for acute variceal bleeding


Management plan
Brief history and clinical examination
No shock mild bleed
Shock moderate to severe massive bleed
Hospitalize
Observe 12–24 hours
Elective investigations plan treatment
Treat shock as outlined in management section A B C
Octreotide/somatostatin
Control
No control
Controlled
Emergency EST/EVL
Plan for investigation to find out source of bleeding EST or EVL as required
No control
Follow up EST/EVL
As per institutional protocol and guidelines
Shunt surgery
Devascularization
TIPS (only in cirrhosis)

Treatment Options Available for Acute Variceal Hemorrhage

1. Endoscopic technique: Sclerotherapy, band ligation
2. Pharmacological intervention:
    i. Vasopressin
    ii. Terlipressin
    iii. Somatostatin.
    iv. Octreotide.
3. Balloon tamponade
   Less preferred if pharmacological intervention or endoscopic sclerotherapy can be applied effectively.
    i. Sengstaken-Blakemore tube
    ii. Minnesota tube.
4. Surgical technique:
    i. Portacaval shunt
    ii. Distal splenorenal shunt
    iii. Devascularization
    iv. TIPS (limited pediatric experience).

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7.3 FULMINANT HEPATIC FAILURE

Jayanta Bandyopadhyay, Jaydeep Choudhury

Fulminant hepatic failure is the development of signs of advanced liver failure such as hepatic encephalopathy which present within 8 weeks of the onset of liver disease in the absence of previous liver disease. The clinical staging of hepatic encephalopathy is shown in Table 7.3.1.

Management

1. Position: Head in the midline raised at an angle of 30°. Neck flexion should be avoided.
2. A, B, C and admit in ICU. Gastroenterologist should be consulted early.
3. It is ideal to go for the minimum investigations.
4. Tubes: One nasogastric tube, two intravenous lines and one indwelling bladder catheter should be in situ.
5. Assess neurological status and level of consciousness. GCS ≤7 need intubation and ventilation.
6. Intravenous fluid: Two-third maintenance fluid with the following additions:
    i. Intravenous glucose solution concentration should be >12 to 15 percent, for which a central line may be required. These children are prone to hypoglycemia, blood sugar must be maintained between 100 and 150 mg/dL.

TABLE 7.3.1

Clinical staging of hepatic encephalopathy

Stage	*Asterixis*	*EEG changes*	*Clinical manifestations*
Stage 1 – Prodrome	Slight	Minimal	Mild intellectual impairment, disturbed sleep wake cycle
Stage 2 – Impending coma	Easily elicited	Generalized slowing of rhythm	Drowsiness, confusion, inappropriate behavior, mood swing
Stage 3 – Stupor	Present if patient is cooperative	Grossly abnormal slowing	Drowsy, unresponsive to verbal commands, markedly confused, delirious
Stage 4 – Coma	Usually absent	Appearance of delta waves, decreased amplitudes	Hyperreflexia, extensor plantar response, unconscious, decerebrate or decorticate response to pain (4A) or absent (4B)

   ii. Protein free or <0.5 g/kg/day and amino acid mixture rich in branched chain amino acid.
   iii. Sodium should be given at 1 to 2 mEq/kg/day. These children are prone to dilutional hyponatremia. Rehydration fluid should be 0.33 to 0.25 percent saline. Serum sodium need to be corrected to >125 mEq/L.
   iv. Potassium should be given at 2 to 3 mEq/kg/day and serum K to be maintained between 3.5 to 4.5 mEq/L.
   v. Calcium should be given as 10 percent calcium gluconate solution 10 mL diluted in intravenous solution.

7. Feeding should be started early with low protein containing feed.
8. Gut clearance:
   i. Lactulose 10 to 15 mL every 2 to 4 hourly till atleast 3 loose stools/day
   ii. Constant nasogastric tube suction to remove any blood from stomach
   iii. Bowel wash and enema with 50 percent magnesium sulphate.
9. Cerebraledema: Positioning, hyperventilation, mannitol.
10. Bleeding manifestations:
    i. Vitamin K 0.2 mg/kg/day (maximum 10 mg/day) injection daily for 3 consecutive days
    ii. Ranitidine 2 to 4 mg/kg/day injection every 12 hourly
    iii. Fresh frozen plasma 10 mL/kg for control of coagulopathy
    iv. Hematocrit >30 percent and platelet count >30,000/cmm to be maintained.
11. Infection: Broad-spectrum antibiotics are indicated to combat nosocomial pneumonia, bacteremia and urinary tract infection. Antifungal agents may be required.
12. Anticonvulsants: Phenytoin is the anticonvulsant of choice. Lorazepam is the preferred sedative, as it is not metabolized through hepatic P450 pathway.
13. Respiratory failure: In stage 3 or 4 encephalopathy and with profound respiratory compromise mechanical ventilation may be needed.
14. Renal failure: Hepatorenal syndrome is a known complication of liver failure in a previously normal kidney. Early peritoneal or hemodialysis is the preferred treatment.
15. Hypotension and shock: Appropriate fluid management with inotropes as required under CVP monitor.
16. Other modalities:
    i. Flumazenil, the benzodiazepine antagonist
    ii. N-acetyl cysteine in paracetamol induced liver damage
    iii. Branched chain amino acid: L-ornithine L-aspartate if serum ammonia is high
    iv. Glucagon.

Follow-up and Monitoring

1. Hourly: Pulse, BP, RR, temperature, GCS, fluid balance with indwelling catheter. Every 4 to 6 hourly blood glucose
2. Every 12 to 24 hourly: Serum electrolytes and renal function
3. Daily: Hemoglobin, PCV, TC, platelet count, coagulation profile
4. Every 2 to 3 days: Liver function test (including glucose, albumin, coagulation, ammonia, bilirubin, ALT, AST, ALK, GGT) depends on clinical situation
5. CT head if suspecting cerebral edema
6. EEG for encephalopathy grading
7. Abdominal ultrasonography.

Temporary Hepatic Support

1. Hemodialysis, hemoperfusion, exchange blood transfusion and plasmapheresis have been tried with mixed results
2. Extracorporeal liver assist device (ELAD)
3. Finally liver transplantation is the treatment.

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8

Renal Emergencies

Jaydeep Choudhury, Saheli Misra, Jayanta Bandyopadhyay

8.1 ACUTE RENAL FAILURE

Jaydeep Choudhury

Assessment of Severity

1. CVS: Blood pressure, low BP may suggest dehydration or shock and high BP signifies renal disease.
2. CNS: Sensorium may be altered in uremia, seizure may be present.
3. Respiratory system: Hyperventilation signifies acidosis.
4. Abdomen: Palpable kidneys suggest hydronephrosis, distended bladder may be due to urinary obstruction.

Determination of Underlying Cause

The typical signs and symptoms of ARF in different situations are summarized in Table 8.1.1. A quick assessment of the child will help in determining the etiology of ARF. The differentiating features of renal and prerenal causes of ARF are shown in Table 8.1.2.

Baseline Investigations

1. Blood biochemistry: Creatinine, serum electrolytes, Ca, Mg, PO_4
2. Full blood count, red cell morphology, differential counts (HUS)
3. Chest X-ray (cardiomegaly, pulmonary edema)
4. Renal ultrasound: If post-renal causes suspected

TABLE 8.1.1

Typical signs and symptoms of ARF in different situations

History/Symptoms	*Signs*	*Diagnosis*
Vomiting, diarrhea, hemorrhage	Dehydration, shock	Gastroenteritis, hypovolemia
Falciparum malaria, G6PD deficiency, snake bite, mismatched blood transfusion	Variable, depending on individual condition	Acute tubular necrosis
Fever	Petechiae, bleeding	Sepsis, DIC
Recent h/o diarrhea Melena/sudden pallor/ seizure		Suspect HUS, confirm with laboratory results
High fever	Flank tenderness	Pyelonephritis
Sore throat / pyoderma	Hypertension/edema	Acute glomerulonephritis
H/O nephrotoxic drugs		Drug induced ARF
Variable urine output	Suprapubic mass	Obstructive uropathy

TABLE 8.1.2

Differentiating features of renal and prerenal causes of ARF

	Prerenal	*Renal*
History/clinical features		
Dehydration, hypovolemia	Yes	No. May be normal or over-hydration
Renal indices		
Specific gravity	> 1020	normal
Urinary sodium	< 20 mEq/L.	>20 mEq/L
Urine/plasma osmolality	>1:3	<1:3
FENa	<1(< 2 in newborn)	>1(>2 in newborn)
Diuretics and fluid challenge	Positive response	No response

5. Renal indices: Urine specific gravity, osmolality, sodium, urea, creatinine, FENa.
   (To differentiate prerenal and intrinsic renal failure).
   Fractional excretion of sodium (FENa)
   Plasma osmolality 275–295, urine osmolality >700 is normal.

Management

1. Normal saline infusion 20 to 30 mL/kg over 30 to 60 minutes to correct hypovolemia

2. Monitoring of vital parameters
3. Measurement of central venous pressure (CVP)
4. Check over hydration and measure urine output (UOP)
5. Electrolytes, diet—protein, salt that need appropriate adjustments and monitoring

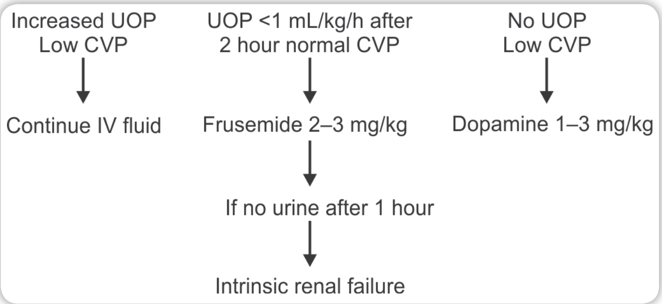
Stepwise management and fluid management of ARF is summarized in Flow charts 8.1.1 and 8.1.2, respectively.

The various complications of acute renal failure and their management are described in Table 8.1.3.

Diet

1. Protein should not be restricted. 0.6 g/kg/day in children and 1g/kg/day in infants
2. Liberal amount of carbohydrate and fat

Flow chart 8.1.1 Stepwise management of ARF



Flow chart 8.1.2 Fluid management of ARF

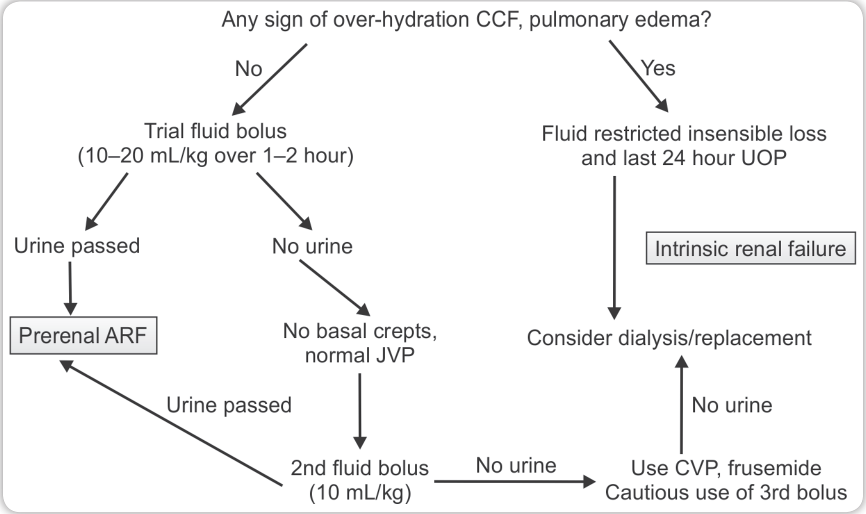


TABLE 8.1.3

Complications of ARF and their management

Complication	*Treatment*	*Remarks*
Fluid overload (Intrinsic renal failure)	5% Dextrose for insensible water loss (400 mL/mt square/day) + 0.45% saline for previous day's urine output.	Other losses accordingly to be monitored and replaced
Pulmonary edema	O_2, frusemide, consider intubation and ventilation	CVP to be monitored
Hyperkalemia (K>5.5 mEq/L)	i. K containing fluid to be avoided ii. Kayexalate (sodium polysterene sulfate) 1 g/kg enema or oral iii. 10% Ca gluconate 0.5–1 mL/kg over 5–10 minutes, to be discontinued if heart rate falls by 20 iv. Salbutamol 5–10 mg nebulization v. 7.5% Sodium bicarbonate 1–3 mEq/kg IV over 30 minutes vi. Dextrose (0.5–1 mg/kg) + Insulin 0.1 unit/kg over 1 hour	Calcium salbutamol stabilizes cell membrane and facilitates K entry into cells
Hypocalcemia (Total Ca <8 mg% or ionized Ca <0.8 mmol%) phosphate	10% Ca gluconate 1–2 ml over 10 minutes with cardiac monitor so that	Latter binds phosphate so that serum Ca X P remains <70 mmol
Premains <70 mmol Hyponatremia (Na <120 mEq/L)	i. Fluid restriction (dilutional hyponatremia) ii. 3% NaCl correct up to 125 mEq/L over 30–60 min (1 mL = 0.5 mEq)	
Metabolic acidosis	If pH <7.2 then, bolus $NaHCO_3$ may be administered (base excess × weight × 0.3) over 30 minutes	
Hypertension	Nifedepine, nitroprusside, diazoxide (avoid betablocker in fluid retention and ACE inhibitor causes potassium retention)	Should be lowered gradually
Anemia	Packed cell transfusion	

3. Vitamin and mineral supplementation.
4. Salt restriction, if there is hypertension.

Infection

Diuretic phase of ARF is characterized by increased urine output, with high blood urea and creatinine. The urine output has to be replaced by 0.45 percent saline infusion.

Indications of Dialysis in ARF

1. Clinical course suggesting prolonged period of oliguria/anuria likely
2. *Uremia*: Altered sensorium, abnormal behavior, seizure, nausea, pericarditis.
3. *Hyperkalemia*: K>6.5 mEq/L or K 5.5 to 6.5 mEq/L with ECG hanges.
4. *Hyponatremia*: Na<120 mEq/L
5. *Fluid overload*: Pulmonary edema, hypertension, CCF resistant to diuretics.
6. *Uncontrolled metabolic acidosis*: pH<7.2 despite $NaHCO_3$.
7. *Hypercatabolic state*: Sepsis, tissue injury, burns.

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8.2 HYPERTENSIVE CRISIS

Saheli Misra

Common Causes

The common causes of hypertension in children in different age groups are shown in Table 8.2.1. The etiology of hypertensive emergencies in children is shown in Table 8.2.2.

Management

1. Hypertensive emergencies should be treated by an intravenous antihypertensive that can produce a controlled reduction in BP, aiming to decrease the pressure by 25 percent or less over the first 8 hours after presentation and then gradually normalizing the BP over 24 to 48 hours.
2. Hypertensive urgencies can be treated by either intravenous or oral antihypertensives, depending on the child's symptomatology.
3. Consider intra-arterial continuous blood pressure monitoring. Continuous parental therapy for BP control should not be administered without continuous monitoring.
4. Provide adequate control of pain and anxiety.
5. When BP is under control with parenteral medications, gradually switch to oral antihypertensives.

   The drugs to treat severe hypertension are shown in Table 8.2.3.

TABLE 8.2.1
Common causes of hypertension in children

Age group	*Cause*
Newborn	Renal vessel thrombosis Renal artery stenosis Congenital renal anomalies Coarctation of the aorta
Early childhood (infancy to 6 years)	Renal parenchymal disease Renovascular disease Coarctation of the aorta
School age (6–10 years)	Renal parenchymal disease Essential hypertension Renovascular disease
Adolescence	Essential hypertension
	Renal parenchymal disease
	Renovascular disease

TABLE 8.2.2

Common causes of pediatric hypertensive emergencies

Renal disease: *(80%)*	
1. *Nephritides:*	Henoch-Schönlein purpura
	Postinfectious glomerulonephritis
	Systemic lupus nephritis
	Rapidly progressive glomerulonephritis
2. *Vascular:*	Hemolytic-uremic syndrome
	Renal artery stenosis and thrombosis
	Renal vein thrombosis
	Sickle cell nephropathy
3. *Congenital malformations:*	Polycystic kidney disease
	Tuberous sclerosis
	Hydronephrosis
	Renal hypoplasia
	Obstructive uropathy
Miscellaneous:	Iatrogenic fluid overload
	Renal failure with fluid overload
	Reflux nephropathy
	Renal tumors
Endocrine:	Pheochromocytoma
	Congenital adrenal hyperplasia
Cardiovascular:	Aortic thrombosis
	Aortic coarctation
	Aortic insufficiency
	Subacute bacterial endocarditis
Drugs:	Corticosteroids
	NSAIDs
	Oral contraceptive pills
	Theophylline
	Phenylephrine

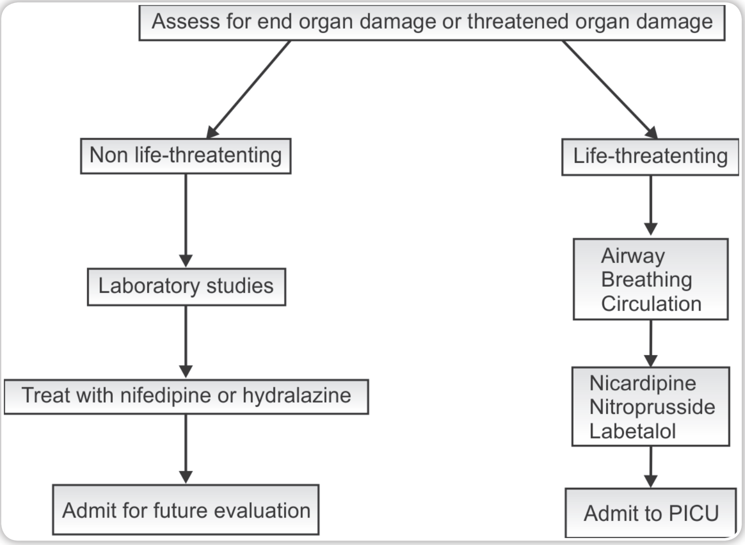
The stepwise management protocol of hypertension in children is shown in Flow chart 8.2.1. Management of hypertensive emergency and urgency are depicted in Flow charts 8.2.2 and 8.2.3, respectively.

TABLE 8.2.3

Drugs to treat severe hypertension

Drug	*Class*	*Dose*	*Route*	*Comments*
Esmolol	β blocker	100–500 µg/kg/min	IV infusion	Very short acting. Constant infusion preferred. May cause profound bradycardia
Hydralazine	Vasodilator	0.2–0.6 µg/kg/min	IV/IM	Should be given every 4 hours when given IV bolus. Recommended dose is lower than FDA label
Labetalol	α and β blocker	Bolus: 0.2–1.0 mg/kg/dose up to 40 mg/dose infusion: 0.25–3.0 mg/kg/h	IV bolus or infusion	Asthma and overt heart failure are relative contradiction
Nicardipine	Calcium channel blocker	1–3 µg/kg/min	IV infusion	May cause reflex tachycardia
Sodium Nitroprusside	Vasodilator	0.53–10 µg/kg/min	IV infusion	Monitor cyanide levels with prolonged (>72 hours) use or in renal failure; or coadminister with sodium thiosulfate

Flow chart 8.2.1 Stepwise management of hypertension



Flow chart 8.2.2 Management of hypertensive emergency

Hypertensive emergency

↓

1. Take a quick history and physical examination
2. Admit to ICU
3. Monitor BP (Preferably arterial line)
4. Place the patient on cardiac monitor
5. Draw blood for CBC, electrolytes, RFT
6. Chest X-ray and ECG
7. Start IV medication

↓

1. The goal for BP reduction is to achieve controlled reduction in BP to minimize the risk of hypoperfusion in cerebral, coronary, and renovascular beds
2. Controlled reduction = Rate of reduction
   1/3 of total amount to be reduced over 6 hours,
   Further 1/3 over 24–36 hours,
   Final 1/3 over 48–72 hours.

↓

There are no absolute recommendations regarding which agent to use.

1. Use the one you are familiar with and most comfortable using
2. Use agent with short half-life
3. Treatment with constant infusion gives steadier, more controlled, and dependable response.

Following drugs may be used:

1. Labetolol

- a. Onset of action: 2–5 minutes
- b. Duration: 2-4 hours
- c. Starting dose: 0.4–1 mg/kg/hr, followed by continuous infusion—0.25–3.0 mg/kg/hour.
- d. Contraindicated in asthma, uncompensated cardiac failure
- e. Recommended for hypertension with high ICP.

2. Sodium nitroprusside

- a. Onset of action: Seconds to 2 minutes
- b. Duration: 1–10 minutes
- c. Starting dose: 0.3–0.5 µg/kg/min (Titrate dose to get the desired effect)
- d. Usual maintenance dose: 3–4 µg/kg/min
- e. Maximum dose: 10 µg/kg/min
- f. Contraindicated in coarctation of aorta, AV shunt
- g. Monitor cyanide level in patient with hepatic dysfunction.

3. Hydralazine

- a. Onset of action in IV use: 5–20 minutes.
- b. Duration of action of IV medication: 2–6 hours.
- c. Bolus doses only
- d. Starting dose: 0.1–0.2 mg/kg/dose IV (maximum 20 mg/dose) every 4–6 hours.
- e. Faster reduction rate (within minutes) would be recommended in cases of pulmonary edema and dissecting aorta.

Flow chart 8.2.3 Management of hypertensive urgency

Hypertensive urgency

↓

1. Admit to the general ward
2. Take history and physical examination
3. IV access is not needed
4. Blood for CBC, electrolytes, renal function test
5. Chest X-ray
6. ECG
7. Start oral medication with one of the following:

1. Captopril:

  a. Initial dose: 0.3–0.5 mg/kg/dose every 6–12 hours
  b. Maximum: 6 mg/kg/day. If a maximum dose reached and the blood pressure still high add a calcium channel blocker.

2. Amlodipine:

  a. Starting dose—0.1 mg/kg/dose every 6–12 hourly
  b. May increase to maximum 0.6 mg/kg/day up to 20 mg/day.

3. Hydralazine:

  a. Starting dose—0.75–1 mg/kg/day every 6–12 hourly
  b. Increase over 3–4 weeks to a maximum dose 7.5 mg/kg/day.

Other oral antihypertensive drugs:

Propranolol:

1–2 mg/kg/day every 6–12 hourly, maximum 8 mg/kg/day. It is the best for pheochromocytoma.

Nifedipine:

It can be used if amlodepine not available
Initial dose is 0.25–0.5 mg/kg/day orally every 6–12 hourly
Maximum dose 3 mg/kg/day up to 180 mg/day
Sublingual route is not recommended
The goal is to reduce BP over longer period—days not hours.

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8.3 HEMATURIA

Jayanta Bandyopadhyay, Jaydeep Choudhury

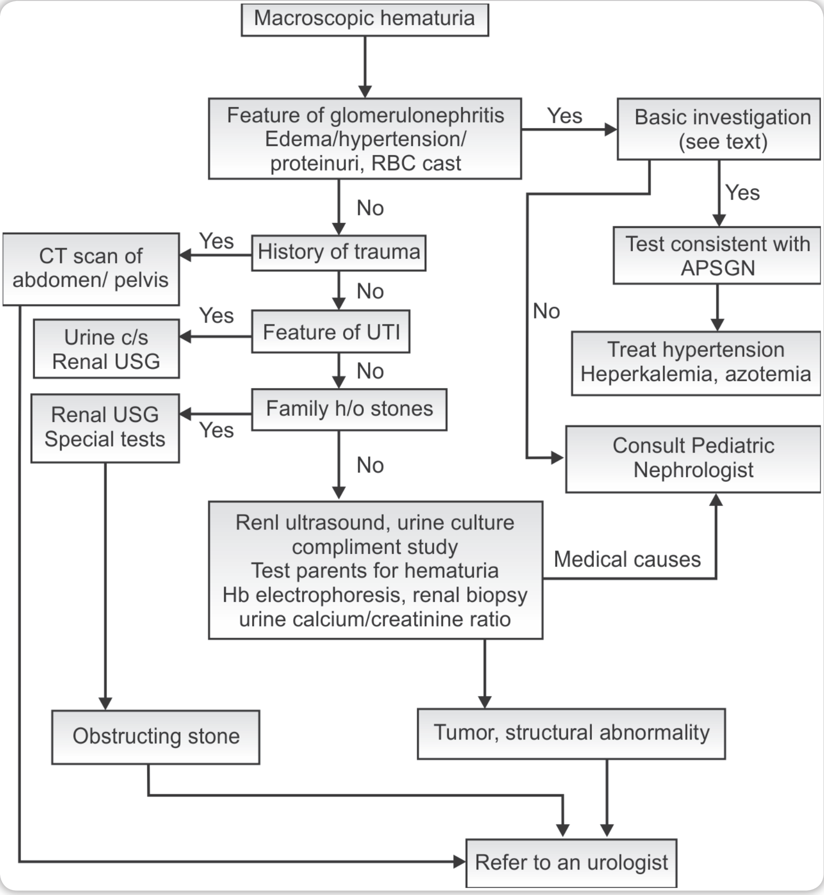
Management

The Algorithm for approach to macroscopic hematuria is shown in Flow chart 8.3.1.

Important Considerations

1. Painful gross hematuria is caused by infection, calculi and urological conditions.

Flow chart 8.3.1 Algorithm for approach to macroscopic hematuria


Macroscopic hematuria
Feature of glomerulonephritis Edema/hypertension/ proteinuri, RBC cast
Yes
Basic investigation (see text)
Yes
Test consistent with APSGN
Treat hypertension Heperkalemia, azotemia
No
Consult Pediatric Nephrologist
No
History of trauma
Yes
CT scan of abdomen/ pelvis
No
Feature of UTI
Yes
Urine c/s Renal USG
No
Family h/o stones
Yes
Renal USG Special tests
No
Renl ultrasound, urine culture compliment study Test parents for hematuria Hb electrophoresis, renal biopsy urine calcium/creatinine ratio
Medical causes
Obstructing stone
Tumor, structural abnormality
Refer to an urologist

2. Most common causes of painless hematuria in children are APSGN and IgA nephropathy.
3. Schistosoma hematobium is often diagnosed by finding an ova in the urine of an unexplained macroscopic hematuria.

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9

Neurological Emergencies

Jayanta Bandyopadhyay, Jaydeep Choudhury

9.1 STATUS EPILEPTICUS

Jayanta Bandyopadhyay, Jaydeep Choudhury

An episode of continuous seizure or intermittent seizures without recovery of consciousness lasting for more than 30 minutes is status epilepticus.

Management

1. A, B, C—airway, breathing and circulation should be taken care of with continuous monitoring
2. Clear airway and suction, insert an airway
3. The child should be kept in lateral prone position to prevent aspiration
4. NG tube insertion to decompress and empty stomach
5. 100 percent O_2 by face mask
6. In case of difficult vascular access an intraosseous route is acceptable. Once a vascular access is established, blood should be collected for sugar, electrolytes, calcium, magnesium, ammonia, sepsis workup and ABG. Blood anticonvulsant levels and toxicology screen may be done in appropriate cases
7. In neonates and infants convulsion due to hypoglycemia or hypocalcemia is common. These should be detected and treated accordingly. Thereby it reduces the chance of administration of several anticonvulsants
8. Bedside capillary blood glucose should be checked early. If blood glucose is less than 40 mg/dL then 10 percent dextrose 2 to 4 mL/kg IV
9. If hypotensive with poor peripheral perfusion treat as in shock
10. Pulse oximetry and blood pressure monitoring should be started early.

Initial Control of Seizure

Short acting benzodiazepines are the preferred first line drugs. Initially before any vascular access is established, diazepam or lorazepam may be given per rectal. The drugs may be given via 6 or 8 Fr feeding tube, 6 cm into the rectum, a 2 mL syringe fitted to the other end of the tube. This may be tried even before the child is transferred to the hospital.

1. Midazolam 0.2 mg/kg intramuscular, intravenous or intraosseous every 10 minutes, 3 does may be given
2. Lorazepam 0.05–0.1 mg/kg/dose mixed in 3 mL 0.9 percent NaCl every 10 minutes, 3 doses may be given
3. Instead of the above two, diazepam may be given 0.3 mg/kg/dose mixed in 3 mL 0.9 percent NaCl every 10 minutes, 3 doses may be given.

Stage I

Midazolam—IM 0.2 mg/kg.
Lorazepam—0.05 to 0.1 mg/kg IV (maximum 4 mg) or
Diazepam—0.3 mg/kg IV (maximum 10 mg) undiluted over 2 minutes.
If IV access could not be established
Diazepam —rectal 0.5 mg/kg, or
↓
If seizure does not stop within 5 to 10 minutes:
Go to stage II

If seizure stops:

Adjust previous antiepileptic medications or start oral anticonvulsants if required. (The decision depends on the likelihood for seizure recurrence).

If patient is shocked or cyanosed with dilated pupils at any stage of management or has been convulsing an hour or more, go straight to stage IV.

Stage II

Repeat midazolam—0.2 mg/kg IV or
Lorazepam—0.05 to 0.1 mg/kg IV (maximum 4 mg) or
Diazepam—0.3 mg/kg IV (maximum 10 mg) undiluted over 2 minutes.
Then start
Phenytoin—15–20 mg/kg (maximum dose 1000 mg) IV infusion at the rate 1 mg/kg/min under ECG monitor. Prepare infusion as 10 mg phenytoin/mL NS or
Fosphenytoin—30 mg/kg IV infusion at the rate 3 mg/kg/min.
Consider pyridoxine—100 mg IV for children <2 years of age.
Start 20 percent mannitol—5 mL/kg over 20 minutes.
If still convulsing 10 minutes after starting phenytoin a 3rd dose of diazepam may be given.

↓

If there is response; continue phenytoin 5 mg/kg/day qevery12 hours
Follow blood level
If no response 5 minutes after the end of phenytoin infusion.

↓

Stage III

Phenobarbitone—loading dose 15 to 20 mg/kg IV, slowly over 10 minutes.
Be prepared for ventilation and if there is response continue maintenance

Phenobarbitone—5 mg/kg/day every 12 hours.
If no response in 5 to 10 minutes after end of infusion or seizure already more than 60 minutes or unstable vital signs.

↓

Stage IV (ICU)

Intubation and ventilation ± muscle relaxant (use short acting muscle relaxant in repeated doses to monitor seizure when EEG monitoring is not available).

Midazolam—0.2 mg/kg IV bolus.
Then infusion 1 to 2 μg/kg/minute titrated up to10 μg/kg/min
(During infusion maintain phenytoin and phenobarbitone at high therapeutic level) or propofol 1 to 2 mg/kg followed by 2–10 mg/kg/h. If seizure is not controlled in 1 to 2 hours induce barbiturate coma.

Thiopentone—2 to 8 mg/kg loading then reduce infusion to 1 to 10 mg/kg/h when needed, titrating for best control.
Monitor for BP, hypoglycemia, electrolytes imbalance, hypocalcemia, acidosis consumptive coagulopathy (PT, APTT) and hyperpyrexia.
Restrict fluid to 60 percent maintenance (unless low BP) and continue treatment for brain edema with mannitol every 6 hours ± dexamethasone (With IV ranitidine).
After stabilization consider CT scan brain and workup for possible causes.
Treat for CNS infection if indicated (LP after brain CT scan).

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9.2 RAPID ONSET LIMB WEAKNESS

Jaydeep Choudhury

The child should be assessed and airway, breathing, circulation should be established first. Optimum posture should be maintained for pain relief, if any. Blood pressure should be checked. If there is any obvious trauma, it should be taken care of.

After stabilization, the detailed course of illness should be obtained along with the pattern and evolution of weakness. The important points in history are the following:

A brief outline of the approach is given in Table 9.2.1. The four common causes of acute onset weakness of lower limbs are poliomyelitis, Guillain-Barré syndrome (GBS), transverse myelitis and traumatic neuritis.

Management

Immediate Management

1. Complete bed rest: Change of posture in bed every 2 to 3 hours. The child should be placed on stomach for short period everyday to avoid the risk of pneumonia
2. Correct positioning of affected limbs: The limbs should be kept in optimum position with pillows and rolled towels. The optimum positions are—hip slight flexion, knee 5^o flexion, ankle 90^o (support against the sole), both legs supported from the lateral side to prevent external rotation
3. Passive movement of the joints: 10 minutes 2 to 3 times a day
4. Warm water fomentation: 10 minutes 2 to 3 times a day
5. Symptomatic treatment for fever and pain
6. No restriction for diet if the patient can take
7. No massage or intramuscular injection
8. If the paralysis progresses, immediate action to be taken
9. Mechanical ventilation may be required in some children with progressive paralysis.

Specific Management

1. GBS: Intravenous immunoglobulin 400 mg/kg daily for 5 days or 1 g/kg for 2 days. Plasmapheresis is the most effective therapy
2. Transverse myelitis: Complete immobilization, care of the bladder. Recovery is usually complete within weeks to months.

TABLE 9.2.1

Approach to rapid onset weakness of lower limbs

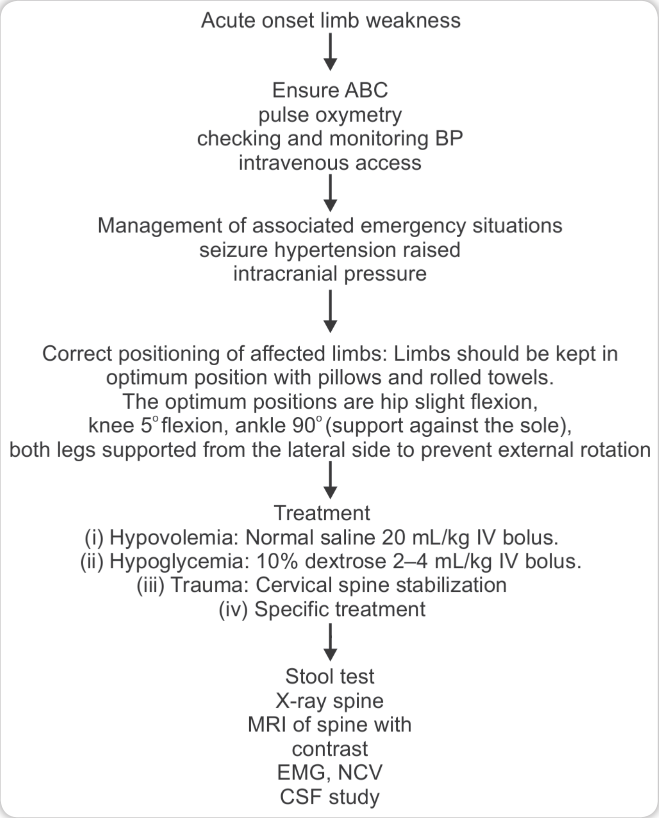
Signs and symptoms	*Poliomyelitis* *Traumatic neuritis*	*GBS*	*Transverse myelitis*
Progression of paralysis	Less than 4 days, max. 7 days From hours to 4 days	From hours to 20 days	From hours to 4 days
Fever at onset	Present Variable	Absent	Absent
Flaccidity	Proximal, asymmetrical Asymmetric limb	Distal, symmetrical	Lower limbs, symmetrical
Muscle tone	Diminished Diminished in limb	Diminished	Diminished in lower limbs
DTRs	Decreased or absent Decreased or absent	Absent	Absent in lower limbs
Sensation	Myalgia, back ache Pain in gluteal region	Cramps, tingling, hyposthesia	Anesthesia of lower limbs, root pain
Cranial nerve	Only when bulbar or bulbospinal Absent	Often present	Absent
Decreased respiration	Only when bulbar or bulbospinal Absent	In ascending paralysis	Absent
Bladder dysfunction	Transient retention Absent	Sometimes	Present
Sequele	Severe asymmetric atrophy Peroneal atrophy	Absent or minimal	Moderate atrophy
CSF: WBC	High Normal	Less than 10	Normal
CSF: Protein	Normal or slight increase Normal	High	Normal or slight increase
NCV (3 weeks)	Normal then slight decrease Abnormal in sciatic nerve	Abnormal demyelination	Normal
EMG (3 weeks)	Abnormal	Normal	Normal

Long-term Management

1. Physical rehabilitation: In poliomyelitis usually there is wasting, weakness of the limbs; proper physiotherapy with exercise like swimming is encouraged. Callipers and braces are used and occasionally wheelchair is required
2. Social rehabilitation
3. Economic rehabilitation.

An approach to the management of acute onset limb weakness is shown in Flow chart 9.2.1.

Flow chart 9.2.1 Management of acute onset limb weakness



Acute onset limb weakness

Ensure ABC
pulse oxymetry
checking and monitoring BP
intravenous access

Management of associated emergency situations
seizure hypertension raised
intracranial pressure

Correct positioning of affected limbs: Limbs should be kept in optimum position with pillows and rolled towels. The optimum positions are hip slight flexion, knee 5° flexion, ankle 90° (support against the sole), both legs supported from the lateral side to prevent external rotation

Treatment
(i) Hypovolemia: Normal saline 20 mL/kg IV bolus.
(ii) Hypoglycemia: 10% dextrose 2–4 mL/kg IV bolus.
(iii) Trauma: Cervical spine stabilization
(iv) Specific treatment

Stool test
X-ray spine
MRI of spine with contrast
EMG, NCV
CSF study

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10

Endocrinal Emergencies

Jaydeep Choudhury, Joshi Anand Karketta, Jayanta Bandyopadhyay

10.1 DIABETIC KETOACIDOSIS

Jaydeep Choudhury

Many children with type 1 insulin dependent diabetes mellitus present for the first time with DKA. As a dictum blood sugar should be routinely checked in any children presenting with coma.

Treatment

Treatment of DKA encompasses several aspects.

Fluid

1. If severe dehydration and shock is present with tachycardia and decreased perfusion, administer 10 to 20 mL/kg plasma expander, like 5 percent albumin or equivalent (0.9% saline) IV rapidly. The same fluid is to be repeated if no obvious improvement is seen.
2. Aim is to replace calculated fluid deficit slowly over first 48 hours + required daily maintenance + continuing losses keeping IV infusion rate as even as possible.
3. *Maintenance (based on weight):* 100 mL/ kg for first 10 kg
   50 mL/kg for next 10 kg
   20 mL/kg for every kg of weight thereafter.
   A 45 kg male requires 1000 + 500 + 500 = 2000 mL maintenance fluid in 24 hours.
4. *Deficit formulae (for 45 kg male):* If estimated dehydration is 5 percent then volume to be replaced is 50 percent of weight in grams. 45 kg male with 5

percent dehydration is 4500/2 = 2250 mL, this should be replaced in 48 hours. Therefore, in first 24 hours body requires maintenance 2000 mL + deficit 1125 mL (½ of 2250 mL)= 3125 mL, which is at the rate of 130 mL/hour.
(*Note:* Recalculate and deduct the fluid from 3125 mL that has been given initially as bolus fluid resuscitation)

5. Nil by mouth until clinical improvement is observed.

Electrolytes

Potassium

1. Add 10 to 20 mEq/L KCl with IV fluid with commencement of rehydration and before insulin has been started unless:
   i. K+ above 5 mEq/L
   ii. Peaked T-wave in ECG
   iii. Previously known to have renal failure.

   In dwelling catheter may be necessary to measure urine output in shocked and comatosed patient.
2. When blood glucose has dropped between 200 to 220 mg/dL change the ongoing fluid to 5 percent dextrose and normal saline with added KCl (50 mL of 50% dextrose to 450 mL of normal saline makes 5% dextrose).

Sodium

1. The measured Na^+ concentration is depressed by the dilutional effect of coexistent hyperglycemia. An approximate corrected sodium can be calculated as:
   Corrected sodium = Sodium measured + 1.6 mEq sodium for every 100 mg glucose above 100 mg.
2. If corrected sodium still shows hypernatremia, correction of dehydration and electrolyte imbalance is advocated over 48 to 72 hours to minimize risk of cerebral edema as an independent glucose hyperosmolar state also exists.

Bicarbonate

1. The role of bicarbonate in correcting acidosis in DKA remains controversial. It should not be given routinely and should be considered with severe acidosis when PH< 7.0 and bicarbonate <5 mEq/L.
   Dose of bicarbonate = base deficit × weight (kg) × 0.3.
   Initially, administer 50 percent of this dose over 1 to 2 hours, added to IV fluids then repeat pH and bicarbonate and reassess.
2. Cardiac monitoring should continue in these patients and the risk of hypokalemia to be kept in mind. KCl needs to be added to the rehydrating fluid if bicarbonate is being administered. These patients should be admitted in ICU.

Insulin

1. Only soluble (short acting) insulin should be used initially. Frequent assessment of insulin dose is mandatory. Ensure that insulin infusion is clearly labeled. Insulin is added to normal saline in a syringe pump so that solution contains 1unit/mL normal saline (add 50 units insulin to 49.5 ml normal saline). This makes further adjustments easy as serious errors occur when calculating dose of insulin.
2. Adjusting insulin dose–Commencement dose = 0.1 unit/kg/hour (>6 years) and 0.05 unit/kg/hour (<6 years).
   Reduce dose once blood glucose < 220 mg/dL (0.05 unit/kg/h if blood glucose 160 to 220 mg/dL.
   Stop insulin when blood glucose is below 160 mg/dL
3. Aim for fall in blood glucose 100 mg/dL/h
4. Occasionally when acidosis is severe or fluids are restricted, the amount of dextrose in IV fluid is increased instead of reducing insulin
5. Once the blood glucose has fallen, acidosis corrected and patient's condition has stabilized, IV insulin infusion should be switched to subcutaneous insulin injections.

Monitoring Initial Progress

1. ***Vital signs:*** For first 24 hours monitor hourly heart rate, respiratory rate, blood pressure, level of consciousness using Glasgow Coma Scale.
   Temperature 4 hourly and daily weights.
2. ***Blood glucose:*** Hourly capillary blood glucose until IV insulin stopped.
3. ***Electrolytes:*** As a minimum, at admission and again in 4 hours but frequency depends on patient's status. Severe cases, electrolytes and even blood gas analysis may be necessary as frequently as 1 to 2 hourly. Acidosis may worsen initially alerting the underlying severity of dehydration. If patient is still poorly perfused, fluid regimen should be revised.
4. ***Fluid balance:*** Strict and careful assessment is mandatory. It may be necessary to catheterize the patient if drowsy, unconscious or severely ill toddlers. Input and output must be interpreted in the context of hydration and blood electrolytes. It may be necessary to increase fluids if large volume of dilute urine is passed and patient is still clinically dry.
5. ***Urine analysis:*** All urine should be tested for ketones. The continued presence of heavy ketonuria suggests either inadequate insulin doses (if blood glucose is high) or inadequate carbohydrate replacement (if blood glucose is low).
6. ***Level of consciousness:*** All patients with DKA should be assessed neurologically using GCS hourly. If score is falling, cerebral edema should be assumed unless another cause is obviously more likely.

Complications During Treatment

1. Cerebral edema
2. Acute gastric dilatation
3. Acute respiratory distress syndrome
4. Venous thrombosis.

Cerebral Edema

It is a life-threatening and unpredictable complication, may relate to overhydration with hypotonic fluids. Cerebral edema can occur suddenly usually between 6 to 12 hours (range 2–24 hours) after commencement of treatment. Mortality and morbidity is high if not treated early. It is most likely to occur at the first presentation in children under 5 years of age.

Warning signs: Failure of sodium level to rise as blood glucose level declines or development of hyponatremia during therapy.

Early signs: Headache, confusion, irritability, decreased consciousness.

Late signs: Bradycardia, hypertension, seizures.

Treatment must be instituted without waiting for CT scan to confirm the diagnosis.

Treatment of Suspected Cerebral Edema

i. Raise the head end of the bed
ii. Administer 1.25 mL/kg of IV 20 percent mannitol (equal to 0.25 g/kg). Repeat in 15 to 20 minutes if the patient has not improved
iii. Intubation and hyperventilation helps reducing raised intracranial tension
iv. Transfer to ICU
v. Reduce IV fluid to maintenance rate
vi. Opinion of Neurologist to be sought and CT scan arranged.

Further Care

Once the nausea and vomiting associated with ketosis have settled, diet can be introduced. This also must correspond to stable metabolic status (normal blood glucose, pH and serum bicarbonate level).

Once the oral intake has been established, change over to subcutaneous insulin is usually possible. Plasma half-life of insulin is only few minutes. Subcutaneous insulin takes 4 to 6 hours to be completely absorbed. It is necessary to continue the insulin infusion at least 1 hour after the first subcutaneous insulin injection.

1. ***Combined subcutaneous insulin:*** A mixture of short acting (soluble) and intermediate acting insulin twice daily (prebreakfast and predinner) is the preferred method. This is because waiting for satisfactory control using

sliding scale with 6 hourly insulin before change to twice daily will delay discharge for some days. The dose used is dependant on patient's weight and insulin requirement over previous 24 hours (IV soluble). In generalour starting total daily doses of 0.4 to 1.0 unit/kg are used.

Prescribe 2/3rd of the total daily dose for morning and 1/3rd for the evening with 2/3rd of each dose as intermediate acting insulin and 1/3rd as short acting insulin.

2. ***Sliding scale subcutaneous insulin:*** Administer soluble (short acting) insulin before breakfast, lunch, evening meal and dinner according to the following scale:

Blood glucose > 350 mg/dL	0.4 unit /kg
Blood glucose > 270–350 mg/dL	0.3 unit/kg
Blood glucose > 180–270 mg/dL	0.2 unit/kg
Blood glucose <180 mg/dL	0.1 unit/kg

Monitoring of blood glucose before main meals and after 3 hours should continue though urine monitoring for ketones and glucose should be stopped once urine is ketone free. Ketosis causes insulin resistance for several days even after insulin treatment has commenced in newly diagnosed type 1 diabetes mellitus. Some initial recovery of natural insulin secretion is also common once treatment is instituted decreasing exogenous insulin requirement rapidly. Frequent hypoglycemic episodes indicate need to reduce dose of insulin.

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10.2 HYPOGLYCEMIA IN NEONATES AND CHILDREN

Joshi Anand Karketta, Jaydeep Choudhury, Jayanta Bandyopadhyay

A blood glucose concentration at/or above 40 mg/dL, preferably in the 54 to 72 mg/dL range represents a more acceptable euglycemic value.

Management

1. Ensure thermoneutral environment
2. Secure an IV access
3. Collect blood for basic investigations
4. If symptomatic give a bolus of 2 to 4 mL/kg IV 10 percent dextrose
5. If asymptomatic do not give bolus as it may cause rebound hypoglycemia
6. Start glucose infusion with 10 precent dextrose at 6 mg/kg/min preferably using an infusion pump
7. Check blood glucose level after 30 minutes
8. If low, increment glucose infusion rate as protocol given below. Following changes in infusion rate check glucose after 30 minutes
9. Calculate separately glucose infusion rate (mg/kg/min) and fluid requirement (mL/kg/day)
10. Need for glucose >12 mg/kg/min or >12.5 percent dextrose to maintain euglycemia suggests refractory (intractable) hypoglycemia. Put a central line to deliver glucose of higher concentration to avoid thrombophlebitis.

Approach to hypoglycemia in neonates is shown in Flow chart 10.2.1.

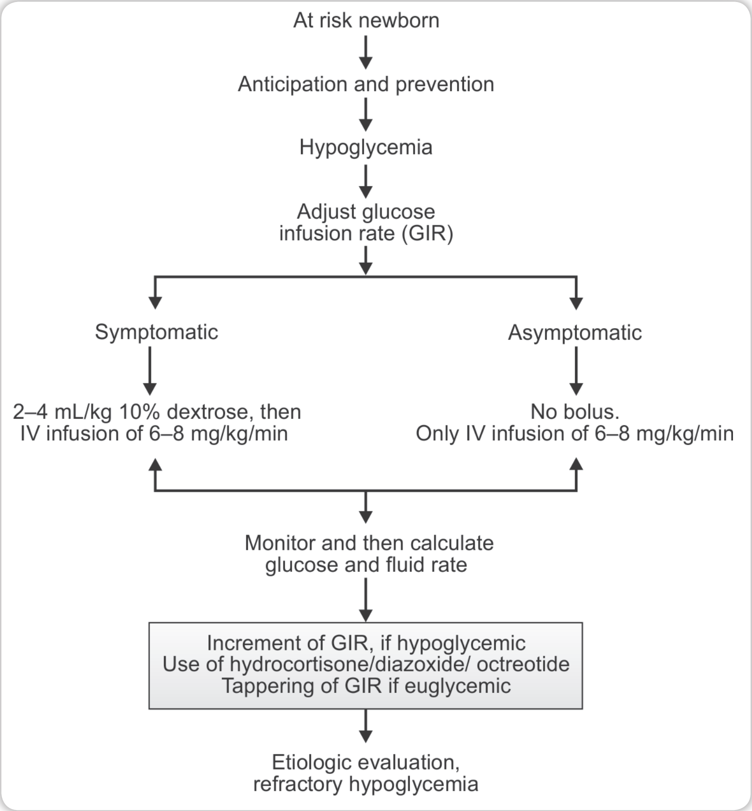
Management for Refractory Hypoglycemia

1. Start hydrocortisone 5 mg/kg/dose, IV, 12 hourly. Collect sample for insulin and cortisol before giving hydrocortisone
2. Glucagon 0.1 mg/kg/ dose, IV or IM as a temporary measure to raise blood glucose or when IV access is difficult
3. Diazoxide l0 mg/kg/day, 8 hourly or octreotide 2 to 4 mg/kg/day 12 hourly
4. Evaluate for underlying etiology and treat underlying specific cause.

Tapering

1. When glucose level crosses 60 mg/dL glucose infusion rate is gradually tapered with decrements of 2 mg/kg/min every 12 hourly, till glucose infusion rate comes down to 6 mg/kg/min with satisfactory blood glucose values more than 50 mg/dL
2. Oral feeds are gradually introduced when glucose infusion is being tapered depending upon infant condition

Flow chart 10.2.1 Approach to hypoglycemia in neonates

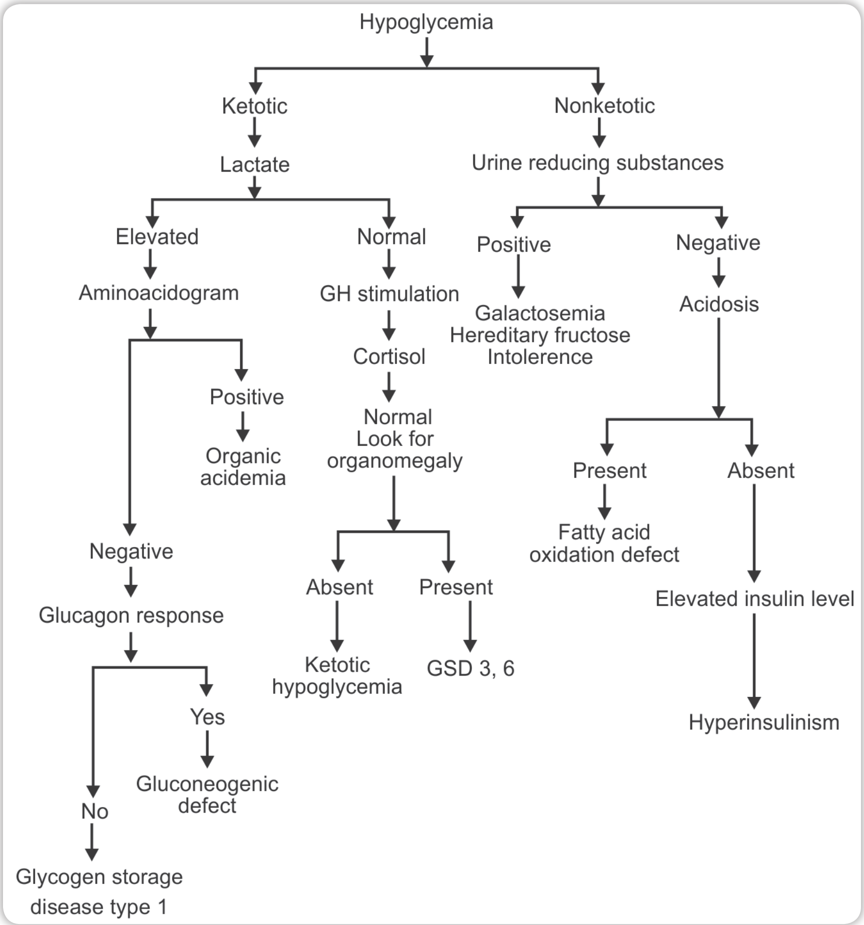

At risk newborn
Anticipation and prevention
Hypoglycemia
Adjust glucose infusion rate (GIR)
Symptomatic
Asymptomatic
2–4 mL/kg 10% dextrose, then IV infusion of 6–8 mg/kg/min
No bolus. Only IV infusion of 6–8 mg/kg/min
Monitor and then calculate glucose and fluid rate
Increment of GIR, if hypoglycemic
Use of hydrocortisone/diazoxide/ octreotide
Tapering of GIR if euglycemic
Etiologic evaluation, refractory hypoglycemia

3. The infant is completely weaned off intravenous infusion when blood glucose values are stable at an infusion of 4 mg/kg/min.
4. Continue hydrocortisone until the infant is stable for 48 hours off IV fluid. This usually takes for 5 to 7 days.
   Diagnostic approach to hypoglycemia in children is shown in Flow chart 10.2.2.

Drugs Causing Hypoglycemia

1. Insulin, oral hypoglycemic agents
2. Antibiotics—sulfonamides, isoniazid, pentamidine, trimethoprim, quinine, ketoconazole
3. Analgesics—phenylbutazone, salicylates, propoxyphene, stanazol
4. Cytotoxic agents—methotrexate, 6 mercaptopurine
5. Anticoagulants—bishydroxycoumarin
6. Psychotropic drugs—ethanol, methanol, lithium haloperidol, tricyclic antidepressants, chlorpromazine, fluoxetine

Flow chart 10.2.2 Diagnostic approach to hypoglycemia in children



7. Insecticides—vapor rodenticide, carbamates, organophosphates
8. Cardiac drugs—disopyramide, propanolol, thiazide diuretics, clofibrate, ACE inhibitors.

Important Considerations

1. Monitor carefully any neonate or infant with a blood glucose concentration less than 50 mg/dL
2. Seizures occur in a minority of babies with hypoglycemia. When they do occur, however, they usually imply long standing hypoglycemia
3. Infants with glycogen storage disease can tolerate very low blood glucose concentrations without having significant symptoms
4. There is a normal dip in blood glucose in first 2 to 4 hours postnatally. The maximum risk for hypoglycemia is in first 24 hours and definitely not after

72 hours, unless it is multifactorial in "at risk group". Monitoring for glucose should be done 4 hourly in first 24 hours or whenever baby is symptomatic.

5. Earliest marker for predicting hypoglycemia in the first few hours of life would be cord blood glucose
6. Do not perform blood glucose determination by reagent strip at temperature below 18°C or above 35°C
7. The glucose level can fall by 14 to 18 mg/dL per hour in a blood sample that awaits analysis
8. Oral glucose or feeding alone are not enough once hypoglycemia has occurred because oral glucose is more likely to stimulate insulin release as compared to intravenous glucose infusion
9. In all other cases of hypoglycemia with the exception of galactosemia and fructose intolerance, ketonemia and ketonuria are present at the time of fasting hypoglycemia.

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11

Environmental Problems

Tapan Kumar Ghosh, Jaydeep Choudhury, Jayanta Bandyopadhyay

11.1 ANIMAL BITES

Tapan Kumar Ghosh, Jaydeep Choudhury

All warm blooded animals can be infected by rabies virus. Human rabies is acquired by bites, licks or scratches of rabid animals. Dogs account for 90 to 96 percent of animal bites in India. The difference of the presenting features in these two types is shown in the following Table 11.1.1.

WHO Classification of Bites

Before starting the treatment of animal bite cases in human being, classification of the exposure is most important. The following Table 11.1.2 shows the WHO classification of animal bites cases.

TABLE 11.1.1

Clinical features in furious and paralytic type of rabies

Furious Type (80%)	*Paralytic Type (20%)*
Tingling/numbness at bite site	Tingling/numbness at bite site
Nonspecific symptoms (Fever, malaise, headache, etc.)	Nonspecific symptoms (Fever, malaise, headache, etc.)
Hydrophobia, aerophobia	Ascending paralysis
Photophobia	Coma
Death in 3–5 days (Cardiac and respiratory failure)	Death in 7–21 days (Cardiac and respiratory failure)

Management

Treatment following an exposure (bite, scratch or lick on broken wounds in skin or directly on mucous membrane, i.e. on oral cavity or on anus by suspected rabid animal) will consist of the following stages:

1. Proper wound management
2. Infiltration of rabies immunoglobulin (RIG) in all category III exposures (vide the Table 11.1.2) rabies
3. Antirabies vaccination with modern cell culture rabies vaccine (CCRV)
4. Antitetanus prophylaxis
5. Supportive treatment with antipyretic/analgesics, local and/or systemic antibiotic as required.

The Steps of Management

STEP I – Wound management

In wound management, the most important steps are the following:

i. Thorough washing of wounds under running tap water for at least 10 minutes with the aim of physical elimination/shedding of the viral loads and application of soap/detergent for chemical treatment and changing the pH of the wounds.

TABLE 11.1.2

Type of contact, exposure and recommended postexposure prophylaxis

Category	*Type of contact*	*Type of* exposure	*Recommended postexposure* prophylaxis
I	Touching or feeding of animals. Licks on intact skin.	None	None, if reliable case history is available
II	Nibbling of uncovered skin. Minor scratches or abrasions without bleeding.	Minor	Wound management + Anti-rabies vaccine
III	Single or multiple transdermal bites or scratches, licks on broken skin. Contamination of mucous membrane with saliva (i.e, licks)	Severe	Wound management + Rabies immunoglobulin + Antirabies vaccine

Note: After carefully assessing the category of exposure, the treating doctor should evaluate the course of action to be taken, based on the following general considerations. He should also keep in the mind that with the presently available safe cell culture rabies vaccines (CCRV), it is always safe to offer treatment rather than withhold in doubtful situations.

ii. Application of disinfectants like povidone iodine, spirit, household antiseptics, etc. to remove the remaining virus particles and prevention of secondary infection.

STEP II – Rabies immunoglobulin (RIG)

Infiltration of bases of wound(s) with rabies immunoglobulin (RIG): Neutralization of the virus and forming a coat around the virus thus obliterating virus entry into the nerve endings.

STEP III – Antirabies vaccination (ARV)

A course of postexposure prophylaxis (PEP) either IM or ID route with modern cell culture ARV (CCRV).

STEP IV – Antitetanus prophylaxis

Administration of tetanus toxoid (TT or Td) and/or tetanus immunoglobulin (TIG) as required.

It is to be noted that in wound management application of irritants, cauterization and suturing, i.e. closing of wounds are to be avoided. If suturing is needed for the purpose of hemostasis, it can be done only after administration of RIG.

Rabies Immunoglobulin (RIG)

The antirabies serum (ARS)/rabies immunoglobulin (RIG) provides passive immunity in the form of readymade antibody to tide over the initial phase of the infection. Antirabies serum or RIG has the property of binding to rabies virus, thereby resulting in neutralization of the virus. Two types of RIGs are available:

1. ***Equine rabies immunoglobulin (ERIG):*** ERIG is of heterologous origin raised by hyperimmunization of horses. Currently manufactured ERIGs are highly purified and enzyme refined. The dose of ERIG is 40 IU per kg body weight of patient. As per WHO guideline skin sensitivity testing is not required prior to injection.
2. ***Human rabies immunoglobulins (HRIG):*** These are prepared from the serum of people hyperimmunized with rabies vaccines. The dose of HRIG is 20 IU per kg body weight (maximum 1500 IU). HRIG does not require any prior sensitivity testing (skin test).

RIG is to be infiltrated as much as possible into and around all the wounds; remaining if any is to be given intramuscularly at a site away from the site of vaccination. All the wounds are to be infiltrated with RIG. After calculation of the dose of RIG if it is seen that RIG is insufficient by volume to infiltrate all the wounds, it is to be diluted with normal saline to make it 2 or 3 times of its volume.

Antirabies Vaccines

Active immunization is achieved by administration of safe and potent CCRVs or purified duck embryo vaccines (PDEV). The dosage schedule of cell culture rabies vaccine (CCRV) is same irrespective of the body weight or age of the children.

Types of Antirabies Vaccines

1. ***Cell culture rabies vaccines (CCRV):***
    i. Human diploid cell vaccine (HDCV)
    ii. Purified chick embryo cell vaccine (PCEC)
    iii. Purified vero cell rabies vaccine (PVRV)
2. ***Purified duck embryo vaccine (PDEV):*** All CCRVs and PDEV used for postexposure prophylaxis (PEP) should have potency (antigen content) greater than 2.5 IU per dose.

Reconstitution and storage: The lyophilized vaccine should be reconstituted with the diluent provided with the vaccine immediately prior to use. In case of unforeseen delay, it should be used within 6 to 8 hours of reconstitution.

Presently liquid human diploid cell (HDCV) vaccine is also available.

Intramuscular (IM) Regimen of Cell Culture Antirabies Vaccine

The regimens for IM administration are described below. But the Essen Schedule is only IM regimen to be practiced in India.

Essen schedule: Five dose intramuscular regimen (1-1-1-1-1): Total five injections, single dose on Days 0, 3, 7, 14 and 28. Day 0 indicates date of first injection**.**

Site of inoculation: The anterolateral thigh region is ideal in infants and younger children whereas the older children can take in deltoid regions safely. Gluteal region is not recommended because the fat present in this region traps the vaccine, retards the absorption of antigen and hence impairs the generation of optimal immune response.

Intradermal (ID) Regimen of Cell Culture Antirabies Vaccine

Intradermal regimens consist of administration of a fraction of intramuscular dose of CCRVs on multiple sites in the layers of dermis of skin. The use of intradermal route leads to considerable savings in terms of total amount of vaccine needed for full pre- or postexposure vaccination, thereby reducing the cost of active immunization. But the regimen will not make any economic meaning when one or two doses are used in private clinics. It is better to be used in antirabies clinics and centers (ARCs) where a handful number of cases of animal bites/scratches will be available.

The following vaccines have been approved by Drug Controller General of India (DCGI) currently for use by intradermal route: (i) Purified chick embryo cell vaccines, (ii) Purified vero cell vaccine.

ID Vaccine Regimen

Updated thai red cross (Updated TRC-ID) schedule (2-2-2-0-2): The most ideal ID schedule. This involves injection of 0.1 mL of reconstituted vaccine per ID site and on two such ID sites per visit (one on each deltoid area, an inch above the insertion of deltoid muscle) on days 0, 3, 7 and 28. The day 0 is the day of first dose administration of IDRV and may not be the day of rabies exposure/animal bite. No vaccine is given on day 14.

Pre-exposure Schedule

As rabies is a cent percent fatal disease and children constitute a special risk for getting the infection, it may be advisable to vaccinate children after they attain the age of 3 years and start playing in the streets and may come in contact with street or pet dogs. This schedule is also practiced in at risk. If such children are exposed to rabies by animal bites, 2 booster doses given on Day 0 and on Day 3 will elicit a rapid and stronger secondary immune response which will neutralize the virus and prevents its ascent to the CNS. There is no need for administration of rabies immunoglobulin in patients who had taken a complete course of preexposure or postexposure course of CCRVs in their previous exposure.

Schedule of Pre-exposure Vaccination

Intramuscular: Three doses of any CCRV (1 mL or 0.5 mL depending on the brand) administered on the anterolateral thigh or deltoid region on days 0, 7 and 28.

Intradermal: The dose (0.1 mL) is same for all vaccine brands and 0.1 mL is administered intradermally over the deltoid on days 0, 7 and 28.

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11.2 SNAKE BITE

Jayanta Bandyopadhyay

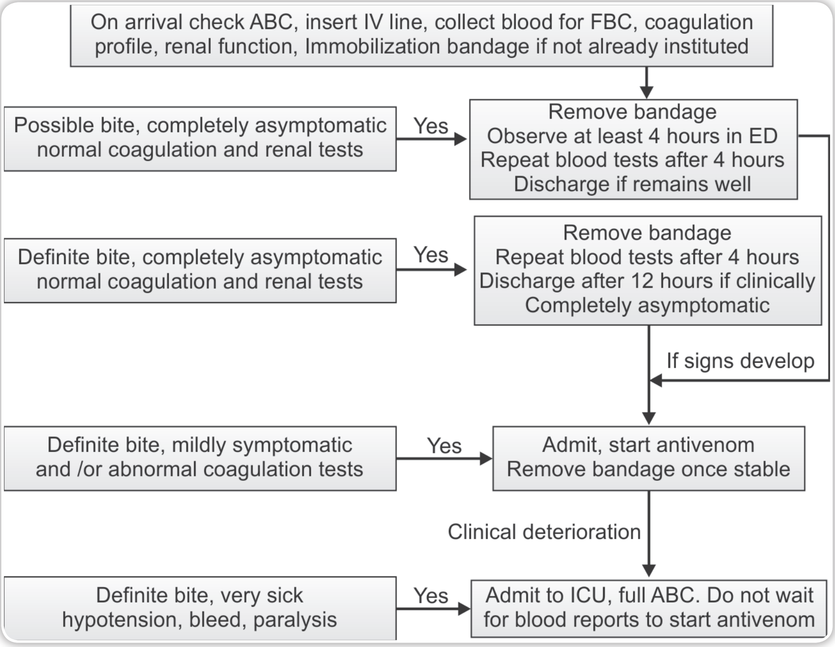
Snake bite is common in rural area. With rapid expansion of urban limits, it is seen in developing urban areas also.

Management

First Aid

1. A wide bandage should be applied to cover the bitten area. The wound should be cleaned with saline.
2. Immobilization of the bitten extremity with a splint or sling.
3. Tetanus prophylaxis as indicated by immunization status.
4. Periods of observation may need to be longer in certain circumstances as shown in Flow chart 11.2.1 management plan.
5. Do not apply suction, arterial tourniquet or incise bitten area.

Flow chart 11.2.1 Snake bite management plan


On arrival check ABC, insert IV line, collect blood for FBC, coagulation profile, renal function, Immobilization bandage if not already instituted
Possible bite, completely asymptomatic normal coagulation and renal tests
Yes
Remove bandage
Observe at least 4 hours in ED
Repeat blood tests after 4 hours
Discharge if remains well
Definite bite, completely asymptomatic normal coagulation and renal tests
Yes
Remove bandage
Repeat blood tests after 4 hours
Discharge after 12 hours if clinically
Completely asymptomatic
If signs develop
Definite bite, mildly symptomatic and /or abnormal coagulation tests
Yes
Admit, start antivenom
Remove bandage once stable
Clinical deterioration
Definite bite, very sick hypotension, bleed, paralysis
Yes
Admit to ICU, full ABC. Do not wait for blood reports to start antivenom

Local Wound Management

1. Wound should be cleaned and left open
2. If swelling or tenderness is present, the proximal edge and time of detection should be marked so that progression can be monitored
3. Similarly the circumference of the limb at the level of edema should be recorded
4. Fasciotomy may be done to prevent compartment syndrome
5. Wound debridement may be required after 3 to 5 days.

Optimize ABC (Airway, Breathing and Circulation)

Assisted ventilation and fluid resuscitation may be required and are important components even before specific treatment is started.

Specific Treatment

Polyvalent antivenin with venoms of four common snakes—cobra, krait and two types of viper are available.

Indications

1. Weakness including ptosis, hypoventilation, bleeding, oliguria, myoglobinuria
2. Collapse, shock or convulsion after confirmed bite
3. Laboratory reports of coagulopathy, deteriorating renal function, myolysis
4. Where laboratory test facilities are not available, 20 minutes whole blood clotting test (20 WBCT) has been advocated. It should be carried out in a clean new and dry test tube. A few milliliter of fresh venous blood is left undisturbed for 20 minutes and then gently tilted. If blood is still in liquid state, it is an evidence of coagulopathy and confirms bite by viperine. Cobras and krait don't cause antihemostatic symptoms.

Do not administer antivenom in patients who only have minor headache, abdominal pain, nausea or vomiting and lymphadenopathy.

Skin Test

It has been abandoned as it has no predictive value.

Pretreatment

Corticosteroid, antihistaminic and ranitidine should be administered before starting antivenin as there is risk of anaphylaxis. Adrenaline, oxygen, fluid and equipments for airway management should be kept ready for emergency. See chapter on anaphylaxis.

Antivenin

Each vial contains 10 mL of antivenin. The total dose necessary in children ranges from 8 to 10 vials depending on the clinical progress and stability of the patient. Maximum 25 vials may be required.

1. Initially 2 vials are diluted in 100 mL normal saline and infused over 1 hour.
2. The rest of the dose is repeated over 4 hours with continuous monitoring. Mainly the development of neurological or hematological manifestations is to be looked for. This infusion may be repeated till the child stabilizes both clinically and blood report wise.
3. After stabilization repeat coagulation profile and renal parameters.
4. Repeat doses of antivenin though rarely required depends on antihemostatic condition and clinical improvement. It should be started 6 hours after finishing the first lot.

If indicated, antivenin should be started as soon as possible and should not be withheld due to late presentation.

Neuroparalysis

Neurotoxin of cobra and krait may produce curare like neuromuscular blockade. Both neuroparalysis and respiratory paralysis may develop.

1. Neogstigmine 0.07 mg/kg intravenous, may be repeated 4 hourly.
2. Atropine 0.02 to 0.50 mg/kg before each dose of neostigmine.
   Constant ECG monitoring should be done.

Supportive Treatment

1. Pain relief.
2. Broad spectrum antibiotic should be started prophylactically to prevent cellulitis, osteomyelitis, septicemia and necrotising fascitis.
3. Renal failure, if develops should be managed appropriately. Sometimes dialysis has to be commenced with caution in the context of coagulopathy.
4. Bleeding manifestations and DIC are life-threatening and should be controlled with blood products and heparin. FFP and platelet will not help unless sufficient antivenom is also given.
5. *Myoglobinuria*: Diagnosed by red or brown colored urine, false positive for occult blood with absent RBC, markedly elevated serum CPK. Forced alkaline diuresis should be instituted and/or early dialysis if rapid elevation of potassium, creatinine occurs.
6. Surgical debridement if required.

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11.3 NEAR DROWNING

Jayanta Bandyopadhyay, Jaydeep Choudhury

Drowning refers to death from asphyxia within first 24 hours of submersion in a liquid. Near drowning is defined as submersion in which survival is greater than 24 hours regardless of morbidity or mortality.

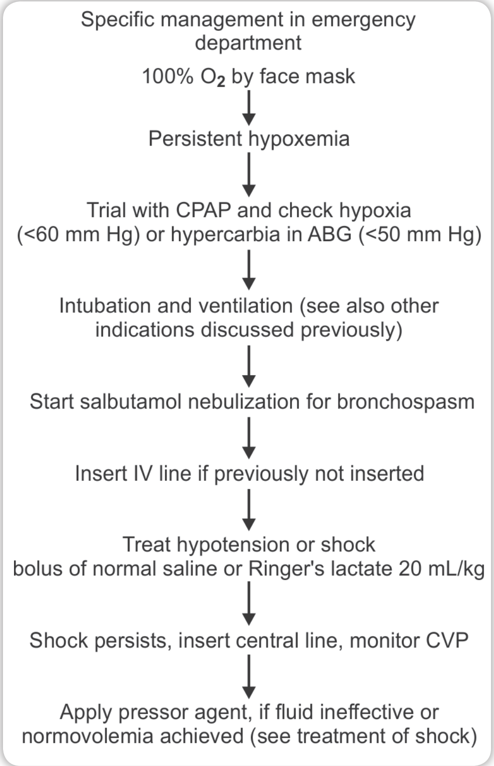
Prehospital Intervention

1. Treatment begins with search, location or removal of patient from water
2. Warming and drying
3. 100 percent O_2 administration
4. Cervical spine immobilization
5. The child should be intubated in the following situations:
    i. Airway not maintainable
    ii. Apnea
    iii. Inadequate respiration or excessive work of breathing
    iv. Cardiopulmonary arrest
    v. Inadequate assisted ventilation
    vi. Prevention of aspiration
    vii. Neurological deterioration
6. Venous or intraosseous access
7. Nasogastric tube, if patient intubated
8. Cardiac monitoring
9. Pulse oximetry, if available
10. Adrenaline if patient is in cardiac arrest.

Emergency Department Management

1. Warming and drying as necessary
2. Immobilize neck if cervical spine injury suspected
3. Vital signs including core temperature
4. Venous access
5. Arterial line or central line if indicated
6. Intubation if indicated
7. Nasogastric tube, Foley's catheter in all unconscious patients
8. Cardiac monitor
9. Pulse oximeters.

The specific management algorithm in emergency department is shown in Flow chart 11.3.1.



Flow chart 11.3.1 Specific management of near drowning in emergency department

Specific management in emergency department
100% O₂ by face mask
Persistent hypoxemia
Trial with CPAP and check hypoxia (<60 mm Hg) or hypercarbia in ABG (<50 mm Hg)
Intubation and ventilation (see also other indications discussed previously)
Start salbutamol nebulization for bronchospasm
Insert IV line if previously not inserted
Treat hypotension or shock bolus of normal saline or Ringer's lactate 20 mL/kg
Shock persists, insert central line, monitor CVP
Apply pressor agent, if fluid ineffective or normovolemia achieved (see treatment of shock)

Hospital Admission Criteria

1. Tachypnea, retractions
2. Hypoxia
3. Abnormal ABG
4. Abnormal Chest X-ray
5. Any neurological impairment.

Hypothermia

See treatment in respective chapter.

Cerebral Edema

1. Elevate head end to 30 degree
2. Maintain PCO_2 25 to 30 mm Hg
3. Minimize procedure which can increase ICP (NG tube, Foley's catheter)

4. Restrict fluid to 50 to 60 percent of maintenance, once vascular stability achieved keeping CVP—8 to 10 mm Hg and urine output 0.5 mL to 1.0 mL/kg/h
5. Mannitol 0.5 g/kg over 30 minutes every 3 to 4 hourly, IV may be used if patient is symptomatic
6. If arrangement is there monitor ICP
7. Induce anesthesia with thiopentone in a dose of 3 to 20 mg/kg IV and maintain 1 to 2 mg/kg/hr while monitoring blood pressure and ICP. Maintain muscle paralysis with pancuromium bromide 0.1 mg/kg IV.

General Measures for Management

1. Correct electrolytes and acid base abnormalities
2. Control seizures with phenytoin 10 to 20 mg/kg IV followed by maintenance 5 mg/kg/day
3. Aspirate stomach content with NG tube
4. Insert Foley’s catheter subsequently
5. Cardiac monitoring for any dysrhythmias
6. No role of steroid has been documented
7. No role of prophylactic antibiotics though empiric antibiotic therapy is a common practice because of chance of streptococcal pneumonia sepsis.

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11.4 BURN

Jaydeep Choudhury

First Aid: To be done at the Site of Burn

1. Flames should be extinguished first. A very effective mode is falling and rolling over the ground. A blanket cover may be applied
2. ***Airway:*** Should be cleaned and patency to be established
3. Clothing and constricting items to be removed. The burns should be covered with water soaked sterile cloth. Recently sustained minor burns should be cooled under running water
4. ***Chemical injury:*** It should be washed off with plain water
5. ***Electrical burns:*** First the source of electrical current should be switched off. If it is not possible then the affected person should be touched with a nonconducting material and separated from the source.

Life Support

1. ***Breathing:*** Humidified oxygen by mask or endotracheal tube
2. ***Circulation:*** Intravenous fluid should be started in all children with >15 percent body surface area (BSA) burn, inhalation injury and electrical injury. Ringer's lactate 10 to 20 mL/kg/hr intravenous should be started
3. Any associated injury should be looked for and evaluated
4. Wounds should be covered
5. A nasogastric tube and a urinary catheter should be kept *in situ*.

Degree of Burn

1° burn: Epidermis only (swelling, erythema and pain)
2° burn: Entire epidermis and variable portion of dermis (vesicle, blister)
3° burn: Entire epidermis and dermis (lack of pain and capillary filling).
1° and 2° burns are called partial thickness burn where as 3^o burn is called full thickness burn.

The calculation of body surface area of different parts of the body in children is detailed in Table: 11.4.1.

Admission Criteria for Pediatric Burns Patients

1. Partial thickness burns involving more than 20 percent BSA
2. Full thickness burns involving more than 5 to 10 percent BSA
3. Suspected smoke inhalation

TABLE 11.4.1
Surface area of different parts of the body in children expressed as percentage of total body surface area (BSA)

Body part	*Newborn*	*3 years*	*6 years*	*12 + years*
Head	18%	15%	12%	6%
Trunk	40%	40%	40%	38%
Arms	16%	16%	16%	18%
Legs	26%	29%	32%	38%

4. Hands, feet, face, perineum and joints burns
5. Burns less than 20 percent BSA in children less than 1 year
6. Comorbidity and other injuries
7. Suspected child abuse.

Fluid Resuscitation

Parkland Formula

1st 24 hours: Ringer's lactate 4 mL/kg/% BSA burned—½ the amount in 1st 8 hours ½ over next 16 hours.

2nd 24 hours: 0.45 percent saline with 5 percent dextrose: ½ of the 1st day's fluid.

5 percent albumin may be used to maintain serum albumin above 2 g/dL
Packed cell transfusion, if Hb is less than 8 to 10 g/dL.

Monitor: Pulse rate, blood pressure and urine output should be at least 1 mL/kg/h.

Injection Td if the child is not already immunized.

Sedation

Should be kept at minimum. The choices are the following:

i. Pethidine 1 mg/kg
ii. Morphine 0.1 mg/kg
iii. Pentazocine 0.1 mg/kg

Infection

i. Wound swab should be collected for culture, blood sample should be taken for culture and sensitivity
ii. Pseudomonas prophylaxis should be started.

Stress Ulcer

Curling's ulcer in the stomach is a known complication. All the burn cases should be given H_2 blocker or other antacids.

Topical Treatment

i. Silver nitrate
ii. Silver sulfadiazine.

Complications

Early: Shock, respiratory tract injury, ARDS, infection, Curling's ulcer, thrombophlebitis, nutritional deficiencies.

Delayed: Post-burn scar, contracture, Marjolin's ulcer.

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12

Poisoning

Jayanta Bandyopadhyay, Jaydeep Choudhury, Joshi Anand Karketta

12.1 GENERAL MANAGEMENT PRINCIPLES AND APPROACH TO UNKNOWN POISONING

Jayanta Bandyopadhyay, Jaydeep Choudhury

Poisoning in children is usually unintentional, involves a single substance found in the household and results in minimal toxicity.

Management

Three basic aspects have to be remembered regarding management of poisoning. When a child presents with unknown, or any poisoning, the child is most likely to recover if ABC is taken care of, timely and adequate gut decontamination is done and complications are effectively managed.

ABC

This is true for any poisoning. Adequate airway and breathing are priority. Circulatory compromise should be managed with fluid resuscitation.

Surface Decontamination

Exposure and contamination of skin requires thorough irrigation with water. Particular attention should be given to hair, fingernails and skin folds. Soap water washing is indicated for hydrocarbons and oily substances. Eye exposure should also be washed with saline water.

Gastrointestinal Decontamination

1. Ideally should be done within 1 hour of ingestion, but should not be denied if the child presents late. Largest possible orogastric tube should be inserted.
2. Patient in left lateral position, normal saline at 15 mL/kg aliquots should be used to wash the stomach contents. The first return sample should be preserved for forensic testing. Lavage should be continued till clear fluid returns.
3. If the patient is obtunded, airway should be protected with an endotracheal tube.
4. Emesis should not be induced in pediatric patients.

Activated charcoal: It should be administered before removal of the lavage tube. It absorbs poison even distal to pylorus and interrupts enterohepatic circulation. Dose is 1 g/kg diluted 1:4 with water or juice. Acids, alkalis, essential oils, cyanide, alcohol, metals like iron, lead, mercury and pesticides like malathion and DDT are not bound by charcoal.

Whole bowel irrigation (WBI): Iso-osmolar polyethylene glycol electrolyte solution (PEGLEC, Tablets, India) is used at a dose of 30 mL/kg/hour for 6–8 hours until the fecal effluents are clear. It flushes out the gastrointestinal tract without causing fluid and electrolyte shifts. This technique is useful in cases of delayed presentation.

Enhanced Elimination

This is reserved for severe toxicity and deterioration in vital function despite adequate supportive therapy.

Alkalinization of urine: Useful for elimination of weak acids like salicylates and barbiturates. The aim is to achieve urine pH of 7.0 to 8.0. Sodium bicarbonate is given 2 mL stat followed by 1 to 2 mL/kg infusion over 6 hours.

Hemodialysis and peritoneal dialysis: Both are effective in removal of absorbed toxins, former is more effective.

Antidotes

Most of poisoning cases respond well to general measures, specific antidotes are rarely required.

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12.2 VARIOUS POISONINGS

Jayanta Bandyopadhyay, Joshi Anand Karketta

Paracetamol

In cases of paracetamol overdose, ideally a 4 hour post-ingestion plasma acetaminophen level should be obtained. It should be plotted on the Rumack-Matthew nomogram to determine potential risk of hepatotoxicity and need for NAC (N-acetyl cysteine) therapy. Rumack-Matthew nomogram is shown in Figure 12.2.1.

Management

1. ***Activated charcoal:*** 1 g/kg immediately if less than 1 hour since ingestion in the proportion 240 mL water/30 g charcoal.
2. ***N-acetyl cysteine (NAC):*** According to FDA/Prescott protocol, NAC therapy should preferably be initiated within 8 hours of ingestion, but is effective when given even more than 24 hours postingestion.

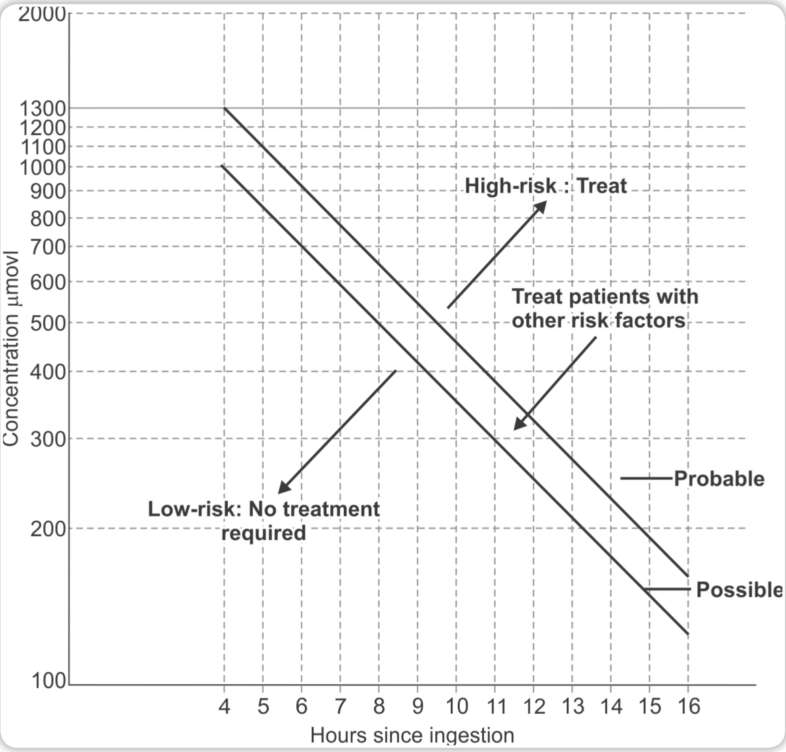


Figure 12.2.1 Rumack-Matthew nomogram

a. *Loading dose:* 150 mg/kg NAC in 200 mL of 5 percent dextrose over 15 minutes,
b. *Second infusion:* 50 mg/kg NAC in 500 mL of 5 percent dextrose over next 4 hours,
c. *Third infusion:* 100 mg/kg NAC in 1000 mL of 5 percent dextrose over next 16 hours.

In young children to avoid excessive amount of free water, prepare a NAC solution of 40 mg/mL (50 mL of 20% NAC + 200 mL of 5% dextrose = 40 mg/mL) and administer as below:

i. *Loading infusion:* 150 mg/kg (3.75 mL/kg) infused over 15 minutes.
ii. *Second infusion:* 50 mg/kg (1.25 mL/kg) over 4 hours (0.31 mL/kg/h).
iii. *Third infusion:* 100 mg/kg (2.5 mL/kg) over 16 hours (0.16 mL/kg/h).

In patients who develop hepatic injury secondary to paracetamol, NAC therapy (0.16 mL/kg/h) should be continued until liver function improves.

Adverse event: Anaphylactoid reactions to NAC may manifest with wheeze and rash. In such situations, infusion should be stopped for 30 minutes and promethazine 0.2 mg/kg IV should be given and infusion should be started at half the previous rate. Increase the rate slowly over time until the desired rate is again reached.

Oral NAC is available as 10 percent and 20 percent solution. It should be diluted to 5 percent solution with fruit juice.

a. *Loading dose:* 140 mg/kg orally as a 5 percent solution,
b. *Maintenance dose:* 70 mg/kg orally as a 5 percent solution every 4 hours for a total of 17 doses. No change in the dose is necessary in patients who have received activated charcoal.

3. ***Hepatic failure:*** Supportive measures for hepatic failure should be started and NAC continued until hepatic function improves.

   Management of paracetamol overdose is shown in Flow chart 12.2.1.

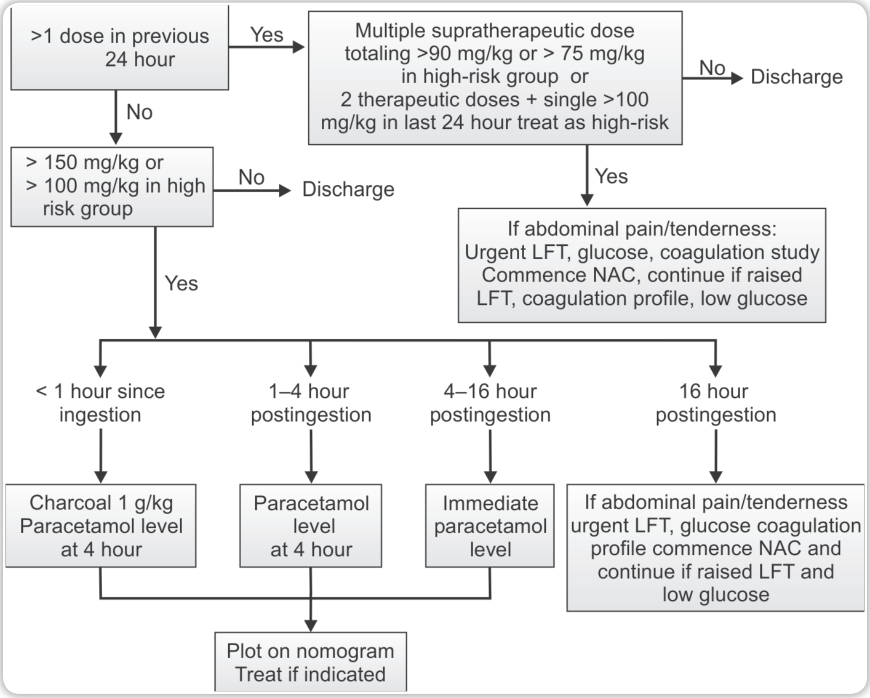
Iron

Management

Iron ingestion management plan is shown in Flow chart 12.2.2.

1. ***Stabilization and support:*** Shock should be treated accordingly. Blood products may be necessary. Control any seizure and protect airway. Initiate baseline investigations.
2. ***Decontamination:***
   i. Repeated gastric lavage may be undertaken to remove all tablets if they are proximal to pylorus. Perform after airway protection is assured.
   ii. If iron tablets are beyond pylorus whole bowel irrigation is the method of choice.
   iii. Few clinicians prefer to perform endoscopic removal of retained tablets in the stomach.

Flow chart 12.2.1 Management plan of paracetamol overdose


>1 dose in previous 24 hour
Yes
Multiple supratherapeutic dose totaling >90 mg/kg or > 75 mg/kg in high-risk group or 2 therapeutic doses + single >100 mg/kg in last 24 hour treat as high-risk
No
Discharge
No
> 150 mg/kg or > 100 mg/kg in high risk group
No
Discharge
Yes
If abdominal pain/tenderness: Urgent LFT, glucose, coagulation study Commence NAC, continue if raised LFT, coagulation profile, low glucose
Yes
< 1 hour since ingestion
1–4 hour postingestion
4–16 hour postingestion
16 hour postingestion
Charcoal 1 g/kg Paracetamol level at 4 hour
Paracetamol level at 4 hour
Immediate paracetamol level
If abdominal pain/tenderness urgent LFT, glucose coagulation profile commence NAC and continue if raised LFT and low glucose
Plot on nomogram Treat if indicated

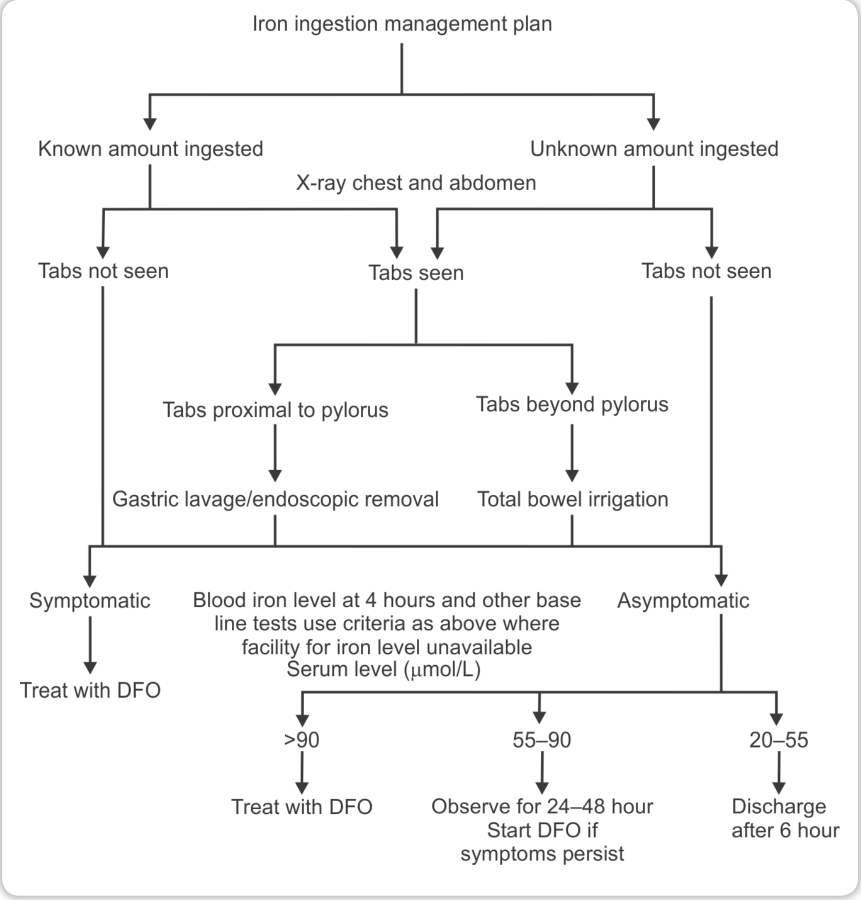
Polyethylene glycol balanced electrolyte solution (PEGLEC) orally or by nasogastric tube is used for whole bowel irrigation. Patient should be seated and/or the head of the bed elevated at least at 45 degree angle.
9 months to 6 years—500 mL/h continue until rectal effluent is clear.
6 to 12 years—1000 mL/h continue until rectal effluent is clear.
>12 years—2 liters initially followed by 1.5 to 2 liters/h continue until rectal effluent is clear.

3. ***Chelation therapy:*** The decision to use desferroxamine (DFO) should be based on clinical status and laboratory value. DFO chelates free iron removing from cellular binding sites and imparts a pink-brown color (Vin-Rose color) to the urine.

   Indication: Serum iron concentration up to 55 to 90 mmol/L (300–500 mg/dL) should be observed for 24 to 48 hours and gastric lavage/bowel irrigation/endoscopic removal are enough if asymptomatic or have nonbloody diarrhea or vomiting.

   IV desferroxamine is urgently needed in patients with hypotension shock, lethargy, coma or convulsion or serum iron concentration >90 mmol/L.
   Dose: 15 mg/kg/h (may be reduced after 2–4 hours).
   Adverse effects: Faster rates, IV boluses or higher doses cause hypotension but infusion rates up to 35 mg/kg/h has been used in children.

Flow chart 12.2.2 Iron ingestion management plan



Discontinuation: End point is adjusted when symptoms subside and urine color is clear. This generally corresponds to 8 to 12 hours in moderate toxicity and 24 hours or longer with severe toxicity.

4. ***Hemodialysis:*** Only useful to remove iron-desferroxamine complex in renal failure.

Organophosphorus Compound

Management

1. Maintain ABCs and secure airway and an IV line
2. Remove all clothes, wash skin with soap and water
3. Administer activated charcoal
4. ***Atropine:*** Antagonizes the muscarinic symptoms. 0.01 mg/kg every 5 to 10 minutes (0.5 mg for toddlers and 2 mg in adolescents) given by IV, oral,

subcutaneous or endotracheal route. Intravenous is the preferred route. Tachycardia is not a contraindication. The end point is satisfactory gas exchange, not pupil size or heart rate

5. ***Pralidoxime (PAM):*** It regenerates cholinesterases and ameliorates the nicotinic symptoms. It must be given with atropine. Most effective if given within 24 to 48 hours. Dose is 20 to 40 mg/kg (maximum 1 g/dose) IV over 30 minutes as 5 percent dextrose in NS. The dose to be repeated after 1 hour and every 3 to 8 hours if symptoms are not relieved or begin continuous infusion of 10 to 20 mg/kg/h.
6. ***Seizures:*** Diazepam 0.2 to 0.5 mg /kg, repeat every 5 minutes as needed. Consider phenobarbitone if uncontrolled
7. ***Acute lung injury:*** Maintain ventilation, assess with ABG frequently.
8. ***Hypotension:*** Isotonic fluid 10 to 20 mL/kg, if persists, dopamine 5 to 20 μg/kg/min titrate to desired response
9. ***Personal protection:*** Rescuer should avoid dermal contact to avoid poisoning themselves.

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13

Foreign Body

Jayanta Bandyopadhyay, Jaydeep Choudhury

13.1 AIRWAY FOREIGN BODY

Jayanta Bandyopadhyay, Jaydeep Choudhury

From management point of view it is necessary to determine the exact anatomic location of the foreign body (FB) like (supraglottic, infraglottic or distal to carina) and also whether the obstruction is complete or incomplete. The typical X-ray features are shown in Figures 13.1.1 and 13.1.2.

Management

The step-by-step management algorithm of airway foreign body is shown in Flow charts 13.1.1 and 13.1.2.

Management of Partial Airway Obstruction

Partial airway obstruction should be approached with extreme caution as one needs to avoid the potential to convert the situation to one of complete obstruction.

Important Consideration

1. If the obstruction is incomplete, move slowly to ensure that incomplete obstruction is not converted into a complete obstruction
2. Call for early help
3. If the obstructing FB is above the vocal cords and can not be removed, immediate cricothyroidotomy is indicated

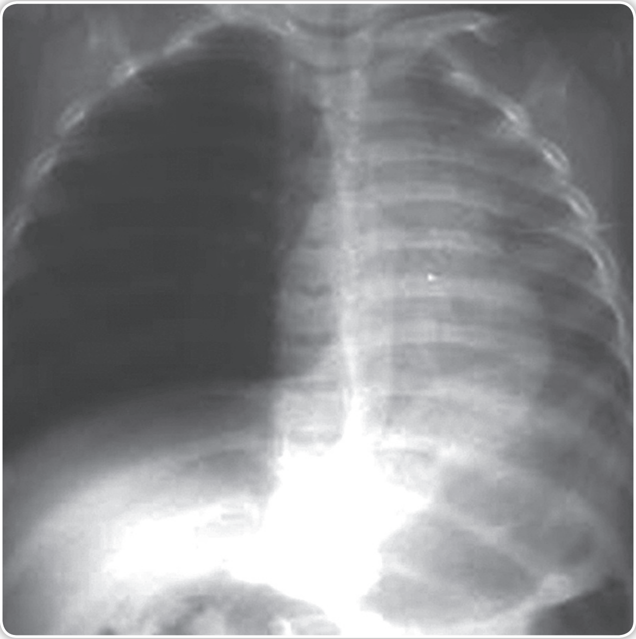


Figure 13.1.1 Expiratory film showing marked air trapping in right lung with right-to-left mediastinal shift

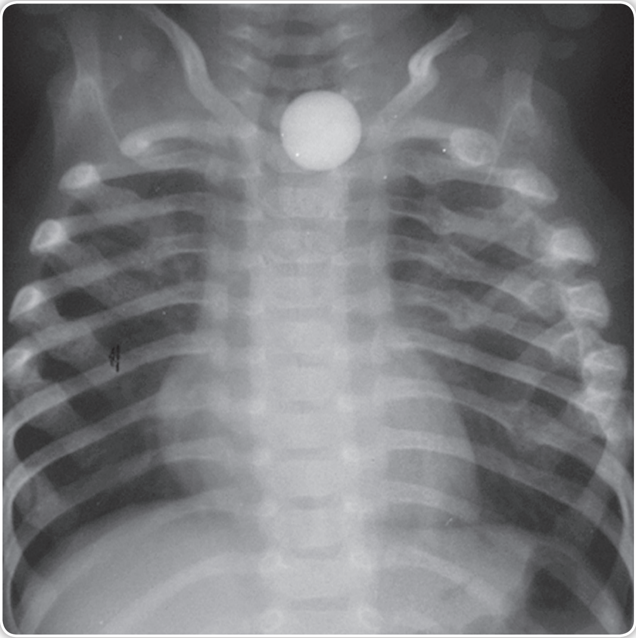
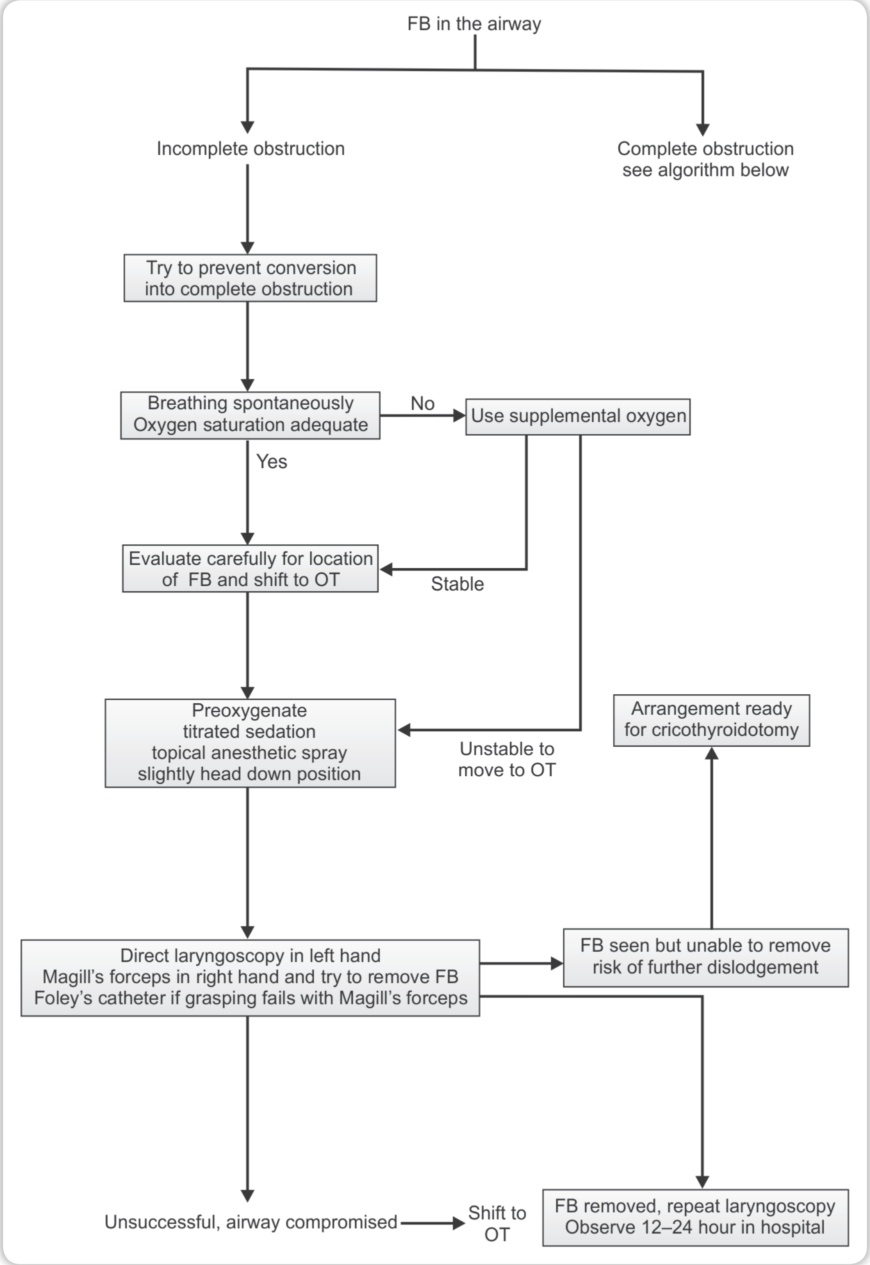
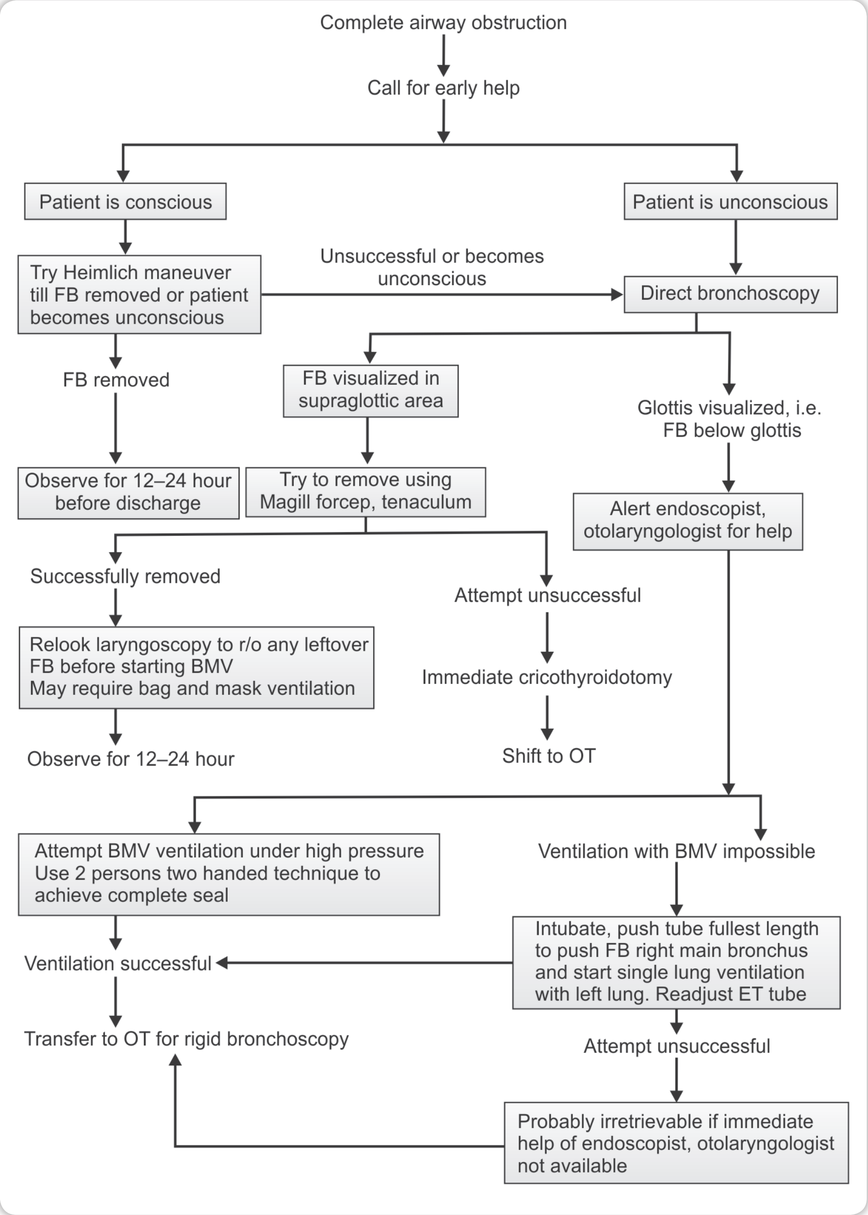


Figure 13.1.2 Radio-opaque foreign body in upper airway

Flow chart 13.1.1 Management of airway foreign body


FB in the airway
Incomplete obstruction
Complete obstruction see algorithm below
Try to prevent conversion into complete obstruction
Breathing spontaneously Oxygen saturation adequate
No
Use supplemental oxygen
Yes
Evaluate carefully for location of FB and shift to OT
Stable
Preoxygenate titrated sedation topical anesthetic spray slightly head down position
Unstable to move to OT
Arrangement ready for cricothyroidotomy
Direct laryngoscopy in left hand Magill's forceps in right hand and try to remove FB Foley's catheter if grasping fails with Magill's forceps
FB seen but unable to remove risk of further dislodgement
Unsuccessful, airway compromised
Shift to OT
FB removed, repeat laryngoscopy Observe 12–24 hour in hospital

Flow chart 13.1.2 Management of complete airway obstruction


Complete airway obstruction
Call for early help
Patient is conscious
Patient is unconscious
Try Heimlich maneuver till FB removed or patient becomes unconscious
Unsuccessful or becomes unconscious
Direct bronchoscopy
FB removed
FB visualized in supraglottic area
Glottis visualized, i.e. FB below glottis
Observe for 12–24 hour before discharge
Try to remove using Magill forcep, tenaculum
Alert endoscopist, otolaryngologist for help
Successfully removed
Attempt unsuccessful
Relook laryngoscopy to r/o any leftover FB before starting BMV
May require bag and mask ventilation
Immediate cricothyroidotomy
Observe for 12–24 hour
Shift to OT
Attempt BMV ventilation under high pressure
Use 2 persons two handed technique to achieve complete seal
Ventilation with BMV impossible
Ventilation successful
Intubate, push tube fullest length to push FB right main bronchus and start single lung ventilation with left lung. Readjust ET tube
Transfer to OT for rigid bronchoscopy
Attempt unsuccessful
Probably irretrievable if immediate help of endoscopist, otolaryngologist not available

4. If the obstructing foreign body is distal to the vocal cords and can not be visualized from above by direct laryngoscopy, cricothyroidotomy will be of no benefit and should not be performed.
5. Heimlich maneuver is a reasonable first-step in any case of complete obstruction.

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13.2 BUTTON BATTERY INGESTION

Jayanta Bandyopadhyay

Button batteries or cells (Figure 13.2.1) are small, smooth and easy to swallow.

Mode of Action

The majority of batteries traverse the gastrointestinal tract uneventfully but complications arise if the battery gets lodged. This occurs most commonly in esophagus. The compositions of various types of batteries are described in Table 13.2.1.

1. ***Corrosive effect:*** Damage to the battery casing may occur in the gastrointestinal tract from acidic attack by gastric contents and/or electrolytic attack due to its own electrical properties, causing leakage of contents. Leakage and/or production of alkali may cause severe caustic injury and local liquefaction. Thus tissue damage may occur from prolonged contact at a single site as the electrical activity of battery cause local short circuit through the tissue, as well as a rise in pH.
2. ***Toxic metal effect:*** Leakage of metal salts may result in systemic toxicity. A single mercury battery contains potentially lethal quantity of mercury for a child.

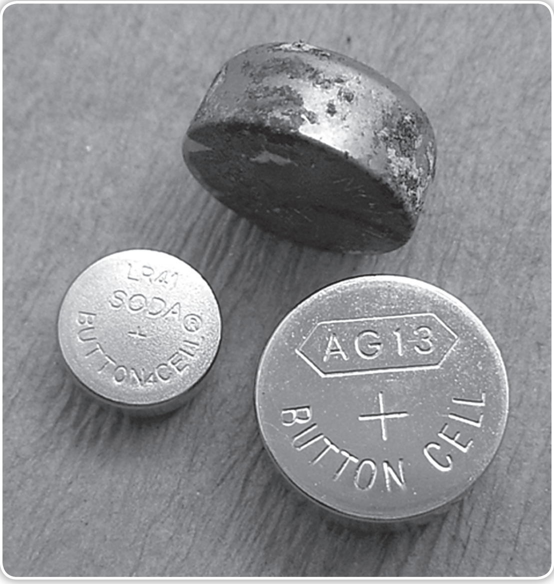


Figure 13.2.1 Button batteries

TABLE 13.2.1
Battery types and composition

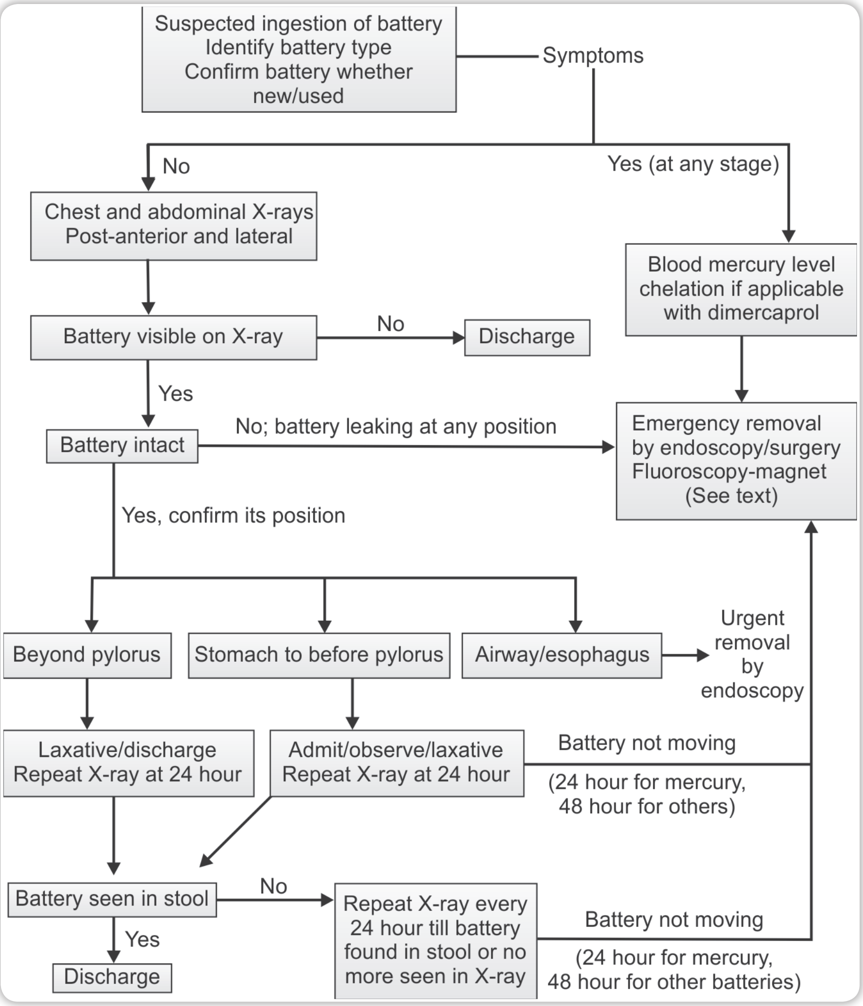
Types	*Main constituent*	*Manganese dioxide*	*Potassium hydroxide*	*Zinc powder amalgum*	*Graphite*	*Zinc oxide*	*Others*
Mercury	Mercuric oxide 40%	5%	7%	14%	1.5%	0.5%	
Silver	Silver oxide 39%	3%	8%	13%		0.15%	Sodium aluminate 0.05%
Alkaline-Manganese	Manganese dioxide	16%	12%	7%	3%	0.5%	Cement 1.5%
Lithium-Manganese	Manganese dioxide	24.8%	1.8%				Propylene carbonate 6%, Lithium perchlorate 1%, Lithium metal anode 2%, Ethylene glycoldimethyl ether 4.3%
Zinc/Air	Zinc metal 32 %	0.3%	10%				Carbon black 2%, Mercury metal 1%

Treatment

Plan of management for button battery ingestion is summarized in Flow chart 13.2.1.

1. Whole bowel irrigation may be considered following ingestion of multiple batteries. The brand and type of battery should be determined from spare battery kept at home.
2. Chest and abdominal X-rays both postero-anterior and lateral to confirm ingestion and position. Button battery can be distinguished from coin ingestion by their lateral view. Coins are usually left to pass spontaneously.

Flow chart 13.2.1 Management plan for button battery ingestion


Suspected ingestion of battery
Identify battery type
Confirm battery whether new/used
Symptoms
No
Yes (at any stage)
Chest and abdominal X-rays
Post-anterior and lateral
Blood mercury level chelation if applicable with dimercaprol
Battery visible on X-ray
No
Discharge
Yes
Battery intact
No; battery leaking at any position
Emergency removal by endoscopy/surgery
Fluoroscopy-magnet
(See text)
Yes, confirm its position
Beyond pylorus
Stomach to before pylorus
Airway/esophagus
Urgent removal by endoscopy
Laxative/discharge
Repeat X-ray at 24 hour
Admit/observe/laxative
Repeat X-ray at 24 hour
Battery not moving
(24 hour for mercury, 48 hour for others)
Battery seen in stool
No
Repeat X-ray every 24 hour till battery found in stool or no more seen in X-ray
Battery not moving
(24 hour for mercury, 48 hour for other batteries)
Yes
Discharge

3. If the battery is still in esophagus, urgent removal by endoscopy is essential.
4. If the battery is intact and in stomach, admit for observation. Osmotic laxatives may be given to reduce transit time in gastrointestinal tract. Abdominal X-ray should be taken every 48 hours (24 hours for mercury or unidentified batteries) until battery is passed in the stools or absent on X-rays.
5. Discharge if battery is intact and beyond pylorus and repeat X-ray after 24 hours.
6. If the battery is in the stomach and shows signs of leakage, it should be removed by endoscopy or by fluoroscopy using magnet.

7. Surgical removal is indicated if endoscopy fails or facility unavailable in the context of signs of leaking, no movement in 48 hours (24 hours for a mercury or unidentified battery) or bloody/tarry stool and /or gastroenteritis.
8. Mercury concentration in blood should be measured in case of mercury battery ingestion.

Emesis and lavage are not recommended, unlikely to evacuate and runs the risk of lodgment into esophagus. Charcoal is also of no benefit as may obscure detection of button cells and discolor stool.

Battery in the Nose or Ear

Extensive corrosive damages have been reported. While swelling, pyrexia, foul smelling discharges are common, and perforation of nasal septum and tympanic membrane are on record. Other complications include hearing loss, facial nerve paralysis.

Battery should be removed immediately and area thoroughly irrigated. Patients with evidence of secondary infection should receive antibiotics.

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14

Psychiatric Behavioral and Social Issues

Jaydeep Choudhury

14.1 PSYCHIATRIC ASSESSMENT FOR NONPSYCHIATRISTS

Jaydeep Choudhury

Mental State Examination

In addition to the neurological examination, following should be noted:

1. *Appearance and behavior*: Attire, cleanliness, self-care, appropriateness of clothing, general health, injuries, nonverbal communication, language, motor function, interaction with interviewer.
2. *Speech*: Rate, quantity, pattern, perseveration, articulation, vocabulary.
3. Ability to read and write.
4. *Thought*: Content, avoided subjects, preoccupations, thought flow, unusual use of language, hallucinations, delusions, obsessions, phobias, paranoia.
5. Apparent intellectual ability.
6. *Mood and affect*: Is the child happy? Sad? Fearful? Angry? Suicidal? Observe the child for appropriate affect—is he/she blunted? Labile? Perplexed? Suspicious?
7. Attitude to family, friends and school. Fantasy life (the child's three magical wishes, the three most wanted people in a deserted island).
8. *Observed play*—content, concentration, distractibility, imagination; significance or symbolism demonstrated?
9. Consider use of mini-mental state examination.

It is important to distinguish between certain medical conditions and those psychiatric conditions which may mimic medical conditions. Symptoms and signs which may indicate both medical and psychiatric conditions are shown in Table 14.1.1.

TABLE 14.1.1

Symptoms and signs which may indicate both medical and psychiatric conditions

Symptom/sign	*Usually suggests*	*May be an indication of*
Acute stridor	Croup	Hysterical stridor (in a child of 7 years or older)
Hyperventilation	Anxiety	Asthma. Could be due to either or both in a child or adolescent with asthma
Acute abdominal pain	Many potential medical and surgical causes	Aerophagy due to anxiety
Recurrent abdominal pain (older child)	Idiopathic abdominal pain of later childhood	Peptic ulceration, renal disease, gall stones
Acute vomiting	Gastroenteritis, other medical or surgical conditions	Somatization of psychological symptoms
Chronic pain	Wide variety of medical problems	Underlying depression
Seizures	Epilepsy	"Pseudoseizures", associated with psychological problems
Amenorrhea	Gynecological cause	Anorexia nervosa
Diffuse muscle pain	Systemic disease	Hyperventilation due to long standing anxiety
Limb paralysis	Acute neurological problem	Conversion disorder
Confusional state	Drug overdose, acute neurological condition	Hysterical fugue
Carpopedal spasm	Hypocalcemia	Hyperventilation from anxiety
Complex partial seizure	Epilepsy variant	Masturbatory habit disorder, in 3–5 year olds
Deafness	Glue ear, congenital deafness	Autism spectrum disorders
Mutism	Organic cause	Selective mutism
Inanition, inability to walk, speak	Major systemic illness	Pervasive refusal disorder

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15

Trauma and Orthopedics

Jayanta Bandyopadhyay, Jaydeep Choudhury, Anirban Chatterjee

15.1 HEAD TRAUMA

Jayanta Bandyopadhyay, Jaydeep Choudhury

Head injury remains the leading cause of accidental deaths in our country in pediatric age group.

Immediate Priority

Establishment of airway, breathing, circulation and cervical spine stability.

Airway

1. Establishment of patent airway with adequate ventilation takes priority
2. High flow oxygen support with bag mask ventilation
3. Jaw thrust and no chin lift to avoid cervical damage
4. Aspiration of gastric content to assist ventilation
5. Suction and maintenance of upper airway (e.g. Guedel's airway)
6. Orogastric (not nasogastric) intubation if fractured cribriform plate is suspected in maxillofacial or forehead trauma
7. Use rapid sequence intubation technique if intubation indicated.

   Atropine (0.02 mg/kg), midazolam (0.1–0.2 mg/kg), suxamethonium (1mg/kg) are perhaps the best choice for less experienced clinician. Alternative is vecuronium (0.1mg/kg) instead of suxamethonium for theoretically more advantageous in raised ICT. Ketamine is contraindicated in raised ICT.

Circulation

1. Treat hypotension urgently and maintain normal blood pressure and cardiac output to prevent cerebral hypoperfusion. Traumatic head injury with shock has a grave prognosis. Attribute other organ trauma to shock than head injury. Significant intracranial blood loss to cause shock is rather uncommon. Hb, PCV and blood group should be checked.
2. Select isotonic nonglucose containing fluid, e.g. normal saline (to prevent cerebral edema). Arrange for colloids, e.g. hemaccel, FFP and blood.

Cervical Spine

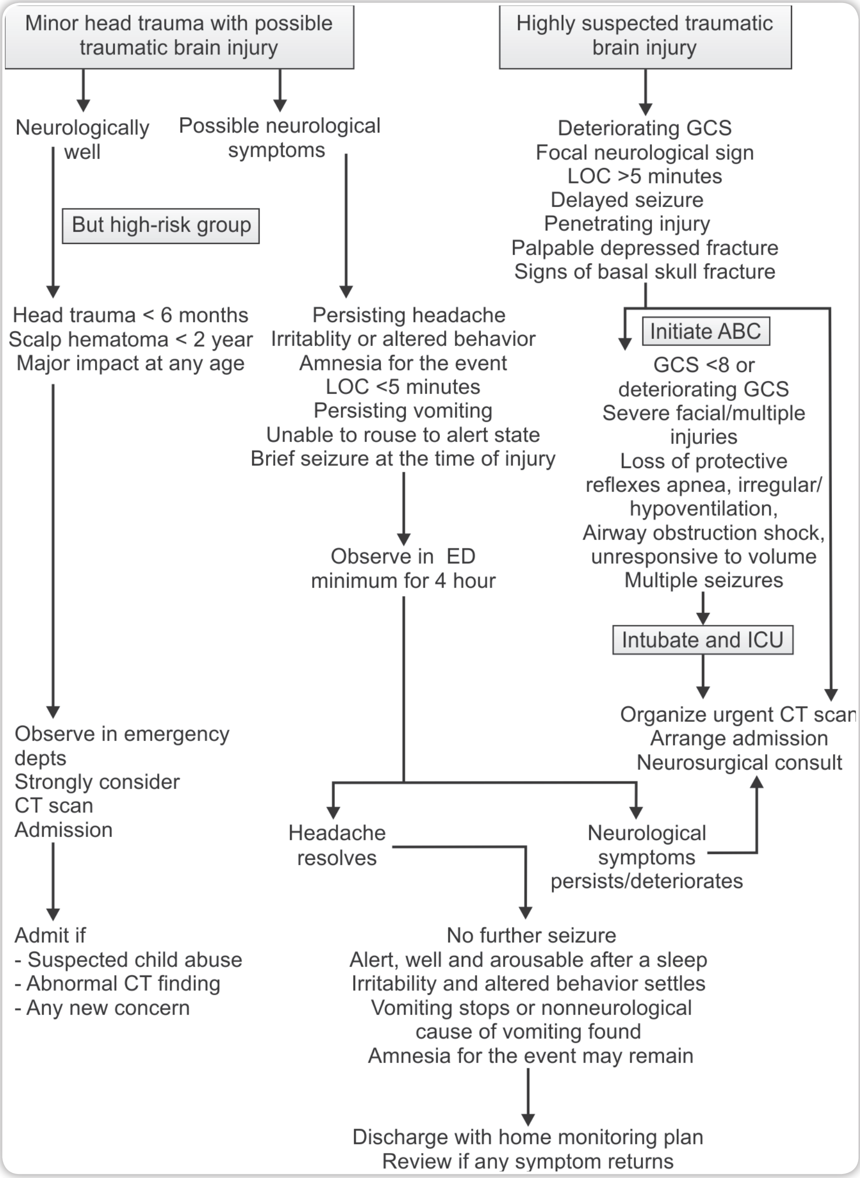
1. Any patient with head trauma must be considered to have spinal trauma and associated injuries until they are excluded. Neutral position should be maintained by use sandbag and semirigid cervical collar. Use log-roll technique to locate tenderness, deformity or to assess cervical spine.
2. Maintain cervical stabilization until radiological clearance.

Assess

1. Level of consciousness: GCS should be monitored. Minor head trauma is associated with score of 13 to 15, moderate 9 to 12 and severe 3 to 8 according to few authors, though this simplification is never absolute and only a guideline.
2. Mechanism of injury: To assess whether there is any major impact.
3. Circumstances of accident: Motor vehicle, sports, diving, fall or blow. Child abuse should also be considered.
4. Chronology: Loss of consciousness (immediate or afterwards) and period of lucidity.
5. Associated findings: Amnesia, disorientation, dizziness, vomiting or nausea, seizure, sensorimotor abnormalities and visual disturbances.
6. Posturing (decerebrate posture or flaccidity have worst prognosis), pupils (size, reaction) eye movement (doll's eye reflex), fundus, sensory responses, refelexes (presence, absence, equality or presence of Babinski's reflex).
7. Other important findings include bulging fontanelle, palpable depression or crepitus of skull, evidence of basilar skull fracture (Battle's sign—ecchymosis posterior to ear, raccoon sign—bilateral black eyes, CSF rhinorrhea, hemotympanum).
8. Simultaneous chest, abdomen, limb, pelvis injury and their severity in relation to priority to their treatment.

Major impact to head includes fall from a height of over 2 meters where there is a reason to suspect that the child has sustained a blow to head or a blow to head as a pedestrian run down or as a motor vehicle passenger in a vehicle traveling at high speed. Head trauma management plan is shown in Flow chart 15.1.1.

Flow chart 15.1.1 Head trauma management plan


Minor head trauma with possible traumatic brain injury
Highly suspected traumatic brain injury
Neurologically well
Possible neurological symptoms
Deteriorating GCS
Focal neurological sign
LOC >5 minutes
Delayed seizure
Penetrating injury
Palpable depressed fracture
Signs of basal skull fracture
But high-risk group
Head trauma < 6 months
Scalp hematoma < 2 year
Major impact at any age
Persisting headache
Irritablity or altered behavior
Amnesia for the event
LOC <5 minutes
Persisting vomiting
Unable to rouse to alert state
Brief seizure at the time of injury
Initiate ABC
GCS <8 or deteriorating GCS
Severe facial/multiple injuries
Loss of protective reflexes apnea, irregular/ hypoventilation,
Airway obstruction shock, unresponsive to volume
Multiple seizures
Observe in ED minimum for 4 hour
Intubate and ICU
Organize urgent CT scan
Arrange admission
Neurosurgical consult
Observe in emergency depts
Strongly consider
CT scan
Admission
Headache resolves
Neurological symptoms persists/deteriorates
Admit if
- Suspected child abuse
- Abnormal CT finding
- Any new concern
No further seizure
Alert, well and arousable after a sleep
Irritability and altered behavior settles
Vomiting stops or nonneurological cause of vomiting found
Amnesia for the event may remain
Discharge with home monitoring plan
Review if any symptom returns

Important Consideration

1. No prospective role of mannitol infusion. Indications of mannitol are evidence of herniation (pupillary dilation, bradycardia), mass effect (hemiparesis). No role of steroid either.
2. Assess neurological status frequently to monitor trends.

3. Pain, struggle causing procedures are detrimental to already raised ICT.
4. The child must be hemodynamically stable before CT scan.
5. Consider prospective anticonvulsants if seizures are prolonged and recurrent which are markers of poor prognosis.
6. Cushing's reflex (bradycardia, hypertension and irregular respiration) is usually a late finding of raised ICP.
7. X-ray study of skull is usually not needed (except suspected depressed fracture with palpable defect, crepitus or history of blunt injury with head of hammer or heel of high heeled shoe) as a linear undisplaced skull fracture is not a critical injury.

Bibliography

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15.2 ACUTE NECK STIFFNESS

Jayanta Bandyopadhyay

Neck stiffness is caused acutely by discomfort resulting from various cervical spine motions. The most common causes being minor trauma, inflammation or infection (usually viral myositis, cervical adenitis, meningitis), oculogyric crisis and congenital muscular torticollis (seen only in infants).

Life-threatening Conditions

The following conditions should be excluded first as these are life threatening:

1. Meningitis
2. Cervical spine injury—fracture or dislocation
3. Retropharyngeal abscess.

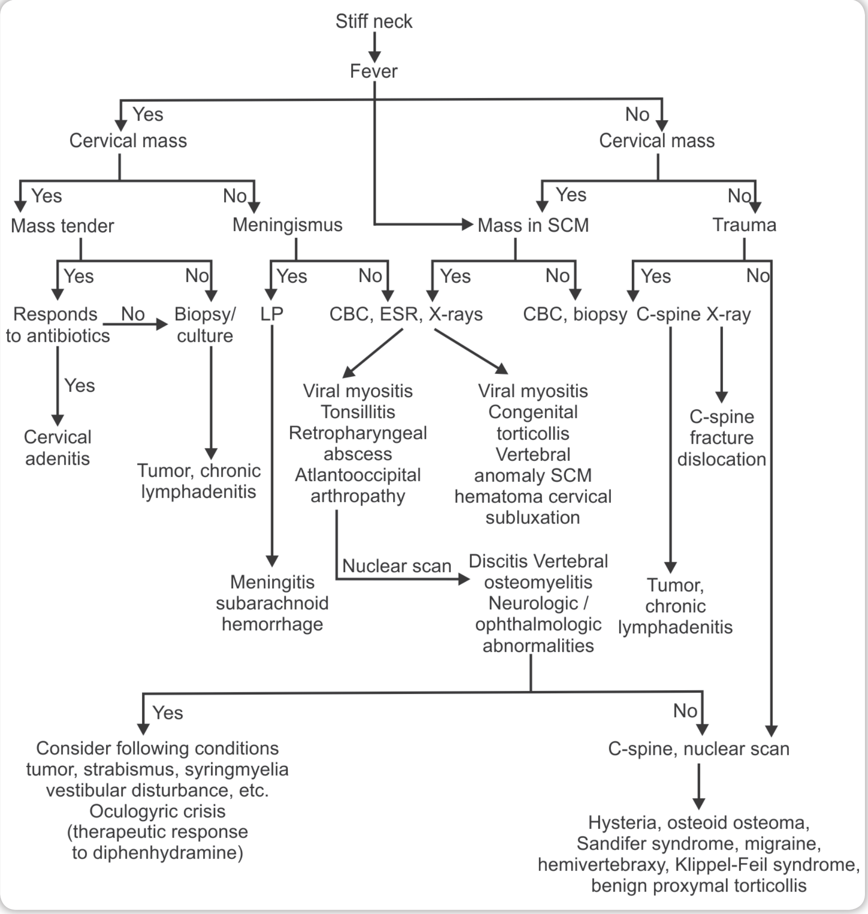
Causes of Acute Stiff Neck

1. ***Trauma:*** Cervical subluxation, muscle injury, hematoma, fracture of clavicle or cervical spine.
2. ***Inflammation or infection:*** Meningitis, cervical adenitis, vertebral osteomyelitis, viral myositis, retropharyngeal abscess, arthropathy associated with pharyngitis.
3. ***Tumor:*** Nerve, muscle or bone tumor with swelling or nerve compression causing pain.
4. ***Oculogyric crisis:*** Usually painless.
5. ***Hysteria:*** Mainly in adolescent girls.
6. ***Down syndrome and acute atlanto-occipital subluxation:*** Children with Down syndrome are more at risk of atlanto-occipital subluxation.

Diagnosis of Acute Stiff Neck

The diagnostic approach to acute neck stiffness is shown in Flow chart 15.2.1.

Flow chart 15.2.1 Diagnostic approach to stiff neck


Stiff neck
Fever
Yes
Cervical mass
No
Cervical mass
Yes
Mass tender
No
Meningismus
Yes
Mass in SCM
No
Trauma
Yes
No
Yes
No
Yes
No
Yes
No
Responds to antibiotics
No
Biopsy/ culture
LP
CBC, ESR, X-rays
CBC, biopsy
C-spine X-ray
Yes
Cervical adenitis
Tumor, chronic lymphadenitis
Viral myositis Tonsillitis Retropharyngeal abscess Atlantooccipital arthropathy
Viral myositis Congenital torticollis Vertebral anomaly SCM hematoma cervical subluxation
C-spine fracture dislocation
Meningitis subarachnoid hemorrhage
Nuclear scan
Discitis Vertebral osteomyelitis Neurologic / ophthalmologic abnormalities
Tumor, chronic lymphadenitis
Yes
No
Consider following conditions tumor, strabismus, syringmyelia vestibular disturbance, etc. Oculogyric crisis (therapeutic response to diphenhydramine)
C-spine, nuclear scan
Hysteria, osteoid osteoma, Sandifer syndrome, migraine, hemivertebraxy, Klippel-Feil syndrome, benign proxymal torticollis

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15.3 ACUTE PAINFUL HIP

Jayanta Bandyopadhyay

The common differential diagnoses are:

1. Transient synovitis
2. Septic arthritis
3. Acute osteomyelitis
4. Slipped upper femoral epiphysis (SUFE)—sudden slip in 30 percent cases.
5. Other less common conditions are epiphyseal-metaphyseal fracture, trochanteric avulsion fractures, cellulitis of the soft tissue around hip.

Transient Synovitis

It is a nonspecific inflammation of the synovium of hip, it is usually of short duration with resolution within 2 to 5 days. Trauma is often coincidental. It is a diagnosis of exclusion.

Management

1. No antibiotics are needed
2. Simple analgesia and rest for 7 to 10 days brings complete resolution
3. Admit and treat as septic arthritis if ESR is high, any sign of bacterial infection, local warmth and significant restriction in joint movement
4. Transient synovitis is usually one-time affair. However, recurrence do occur and then one should consider Legg-Perthes disease (2% of cases).

Septic Arthritis

Infection of the joint capsule by hematogenous origin or due to spread from a neighboring bone lesion caused by Group A streptococcus, *E. coli* (neonates) *Hemophilus influenzae* (below 2 years and nonimmunized), *Staphylococcus aureus* (older children) and salmonella (in sickle cell anemia).

Management

1. Early surgical drainage by arthrotomy or arthroscopy will decrease the risk of late degenerative arthritis.
2. Also admit for IV antibiotics (flucloxacillin with cefotaxime) with upper limit of dosage. Appropriate changes are made according to the bacteriological reports and antibiotics changed to oral (except neonates and infants) once response to treatment is evident. If treatment is commenced early, 3 weeks treatment is sufficient.

Acute Osteomyelitis

An inflammatory process with nidus in the metaphysis due to hematogenous spread by Group A streptococcus, *E. coli* (neonates) *Hemophilus influenzae* (below 2 years and nonimmunized), *Staphylococcus aureus* (older children and commonest) and salmonella (in sickle cell anemia). Trauma to metaphysis enables infection to establish in the tortuous blood vessels. It may then track to the surface as periosteal abscess or to epiphysis in infants. Moreover it may spread simultaneously as septic arthritis as joint capsule in infants insert low on the femoral neck.

Management

1. Admit for IV antibiotics (flucloxacillin with cefotaxime) with upper limit of dosage.
2. Appropriate changes are made according to the bacteriological reports and antibiotics changed to oral (except neonates and infants) once response to treatment is evident (afebrile, local swelling and tenderness subsides and CRP decreases). Uncomplicated cases, 3 to 4 weeks treatment is sufficient whereas bone changes in X-ray require more prolonged treatment (5–6 weeks).
3. Surgery is indicated when collection of pus develops—intraosseous, subperiosteal, intra-articular that means when diagnosis has been delayed. Antibiotics are not effective in the presence of pus and failure to drain leads to chronic osteomyelitis.

Slipped Upper Femoral Epiphysis (SUFE)

It is a condition of late childhood, male: female 4:1. Females 11 to 14 years and male 13 to 16 years mostly affected. Two-thirds of them are overweight and sexually immature, 50 percent have history of trauma. Systemic conditions associated with SUFE include hypothyroidism, panhypopituitarism, hypogonadism, ricket and exposure to radiation. Only in 30 percent slip occurs suddenly, though in 70 percent slip is gradual.

Management

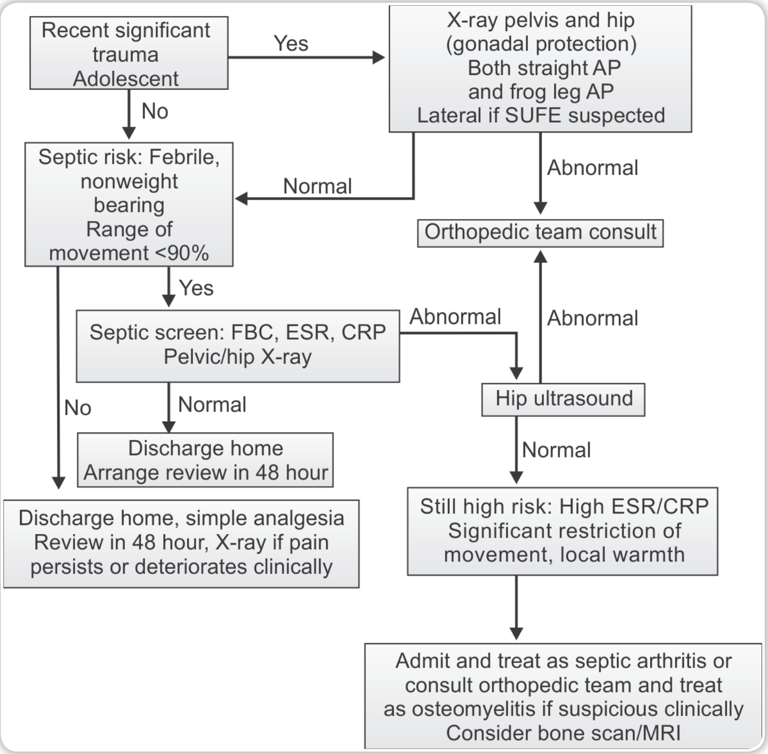
Regarded as orthopedic emergency, admitted for prompt surgery.

1. Modest displacement (<1/3) is percutaneously pinned to stabilize the epiphysis and precipitate closure of proximal growth plate.
2. Greater displacement (>1/3) requires reduction and pinning.

Other Conditions

Depends on the history of trauma and X-ray findings. Cellulitis of the soft tissue around hip may mimic septic arthritis or osteomyelitis. Most often cellulitis

Flow chart 15.3.1 Management plan of acute painful hip


Recent significant trauma Adolescent
Yes
X-ray pelvis and hip (gonadal protection) Both straight AP and frog leg AP Lateral if SUFE suspected
No
Septic risk: Febrile, nonweight bearing Range of movement <90%
Normal
Abnormal
Orthopedic team consult
Yes
Septic screen: FBC, ESR, CRP Pelvic/hip X-ray
Abnormal
Abnormal
Hip ultrasound
No
Normal
Discharge home Arrange review in 48 hour
Normal
Discharge home, simple analgesia Review in 48 hour, X-ray if pain persists or deteriorates clinically
Still high risk: High ESR/CRP Significant restriction of movement, local warmth
Admit and treat as septic arthritis or consult orthopedic team and treat as osteomyelitis if suspicious clinically Consider bone scan/MRI

is due to inflammation of the inguinal lymphnodes and can be differentiated from others by ultrasound by showing increased blood flow with inflammatory adenopathy in cellulitis against joint fluid in septic arthritis.

The overall management plan of acute painful hip is shown in Flow chart 15.3.1.

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15.4 SPINAL CORD INJURY AND SPINAL CORD INJURY WITHOUT RADIOGRAPHIC ABNORMALITY (SCIWORA)

Jayanta Bandyopadhyay, Jaydeep Choudhury

Injuries to the spines and spinal cord are less in children than adults. Children account for up to 10 percent of all spinal injuries. The common causes of spine and spinal cord injuries in children are motor vehicle crashes, falls, diving accidents, sport injuries and occasionally nonaccidental injury.

Initial Assessment

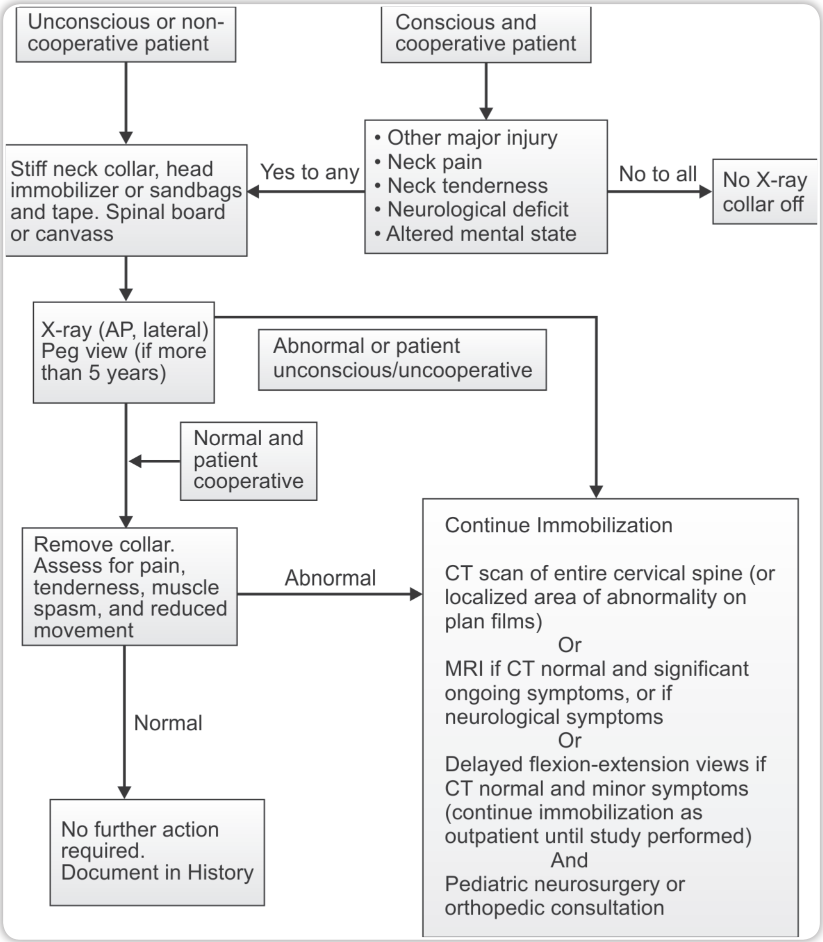
All patients with significant trauma should be assumed to have a spine or spinal cord injury and appropriate precautions must be taken to prevent further exacerbating any possible injury. The initial assessment of patients with potential spine or spinal cord injury should be directed at the airway, breathing and circulation, in line with trauma resuscitation guidelines. The patient should be stabilized and a thorough secondary survey performed. At this point, all possible spine and spinal cord injuries should be identified. The lateral cervical spine X-ray will be done at this stage in patients with major trauma. Thorough radiological assessment of the injuries should be completed once resuscitation and stabilization and the secondary survey have all been accomplished.

Management

Management must start with care of the airway, breathing and circulation. Only once these areas have been stabilized should management of the spine proceed. However, while the patient is being stabilized the spine should be maintained in alignment and patient moved via log rolling. A thorough assessment and investigation of the abdomen and chest is mandatory for all patients with significant thoracic and upper lumbar spine injuries, and injuries to the pelvic must not be forgotten with lumbar spine injuries. As many of these injuries are associated with intra-abdominal injuries an ileus is common and nasogastric or orogastric tube should be inserted. Consultation with a pediatric orthopedic surgeon or neurosurgeon should be sought for definitive care of the injury. Surgical stabilization is usually required for unstable fractures and those fractures associated with neurological injury.

Management of the potentially injured cervical spine is shown in Flow chart 15.4.1.

Flow chart 15.4.1 Guideline to management of the potentially injured cervical spine


Unconscious or non-cooperative patient
Conscious and cooperative patient
• Other major injury
• Neck pain
• Neck tenderness
• Neurological deficit
• Altered mental state
Yes to any
No to all
No X-ray collar off
Stiff neck collar, head immobilizer or sandbags and tape. Spinal board or canvass
X-ray (AP, lateral) Peg view (if more than 5 years)
Abnormal or patient unconscious/uncooperative
Normal and patient cooperative
Remove collar. Assess for pain, tenderness, muscle spasm, and reduced movement
Abnormal
Normal
Continue Immobilization
CT scan of entire cervical spine (or localized area of abnormality on plan films)
Or
MRI if CT normal and significant ongoing symptoms, or if neurological symptoms
Or
Delayed flexion-extension views if CT normal and minor symptoms (continue immobilization as outpatient until study performed)
And
Pediatric neurosurgery or orthopedic consultation
No further action required. Document in History

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15.5 BLUNT TRAUMA

Jayanta Bandyopadhyay, Jaydeep Choudhury

Motor vehicle collisions, automobile versus pedestrian accidents, bicycle injuries and falls are associated with the greatest risk of blunt trauma. Childhood sports and recreational activities can also sometimes produce blunt trauma.

Basic Principles of Trauma Management

1. Do not waste time—even a well looking child may deteriorate rapidly.
2. Occult injury should be suspected—internal injury may not be manifested always.
3. Each child should be approached systematically.

Management

1. Urgent investigations: CBC, LFT, X-rays and urinalysis.
2. Fluid resuscitation: Particularly in unstable children. Normal saline or Ringer's lactate 20 mL/kg initially.
3. Blood: Continued hemodynamic instability even after 2 boluses should be treated with 10 mL/kg packed RBC.
4. CT scan: As a part of initial assessment if the child is stable and facility is available. Unstable children should be stabilized first before going for CT scan.
5. Surgical referral.

Abdominal Trauma

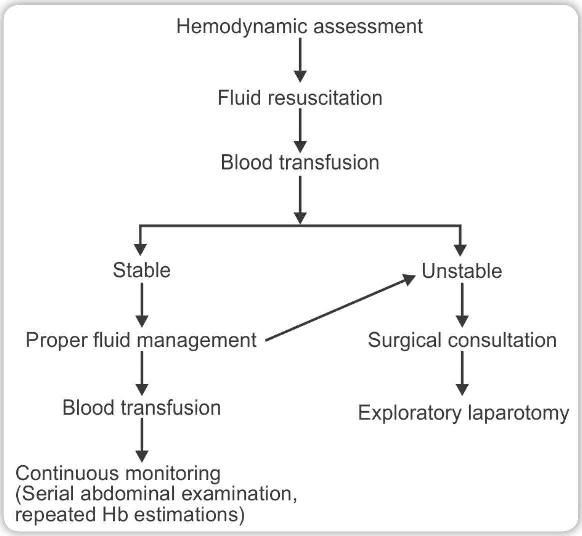
There are some unique features in children which make them more vulnerable to abdominal injuries. The rib cage does not extend as far down as in adults, the ribs are more compliant and the abdominal wall musculature is thinner. Consequently the upper abdominal viscera are more at risk of injury.

Liver Trauma

Spleen and liver are the two organs commonly injured by blunt trauma in children.

Abdominal CT scan, particularly contrast CT scan is helpful in determining the extent of injury. Hemodynamic assessment is most important in any child with trauma. Management of liver trauma is outlined in Flow chart 15.5.1.

Flow chart 15.5.1 Management of liver trauma



Splenic Trauma

Splenic trauma should be suspected in children with left upper quadrant tenderness, left lower rib fractures or evidence left lower abdominal or chest contusion. The spleen is a commonly injured organ in blunt abdominal trauma. CT scan finding of splenic rupture is shown in Figure 15.5.1 and management of splenic trauma is shown in Flow chart 15.5.2.

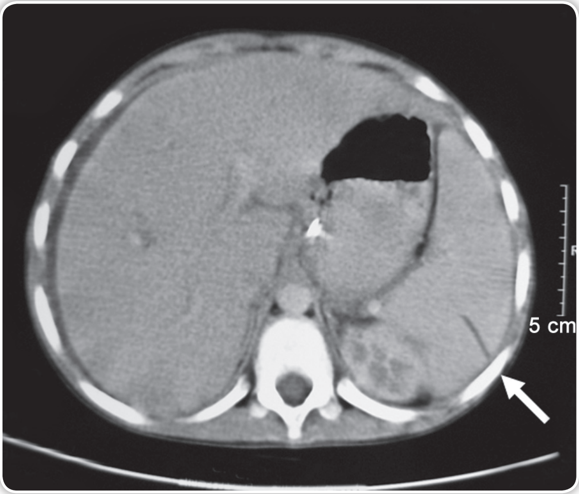
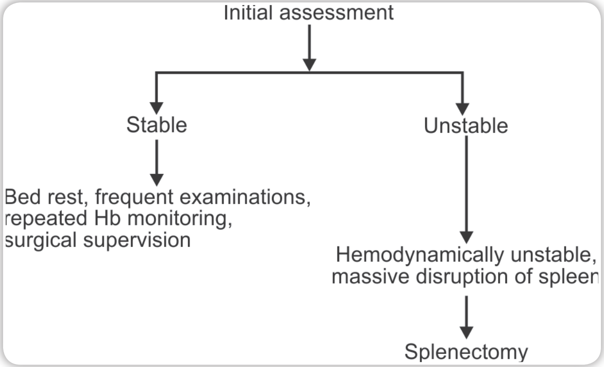


Figure 15.5.1 CT scan showing splenic rupture

Flow chart 15.5.2 Management of splenic trauma


Initial assessment
Stable
Unstable
Bed rest, frequent examinations, repeated Hb monitoring, surgical supervision
Hemodynamically unstable, massive disruption of spleen
Splenectomy

Pancreatic Trauma

Though pancreatic trauma is rare in children but it should be suspected in children who falls on the horizontal handle bar of a bicycle and sustains a crush injury. It is often very difficult to diagnose clinically. CT scan abdomen may be helpful.

Renal Trauma

Most renal trauma is the result of motor vehicle accident. Presence of visible or microscopic hematuria following abdominal trauma should raise the suspicion of renal injury. Most renal trauma heals with conservative management. The overall management is similar to liver trauma.

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15.6 FRACTURES: INITIAL STABILIZATION AND MANAGEMENT

Anirban Chatterjee

Musculoskeletal injuries in children are fairly common accounting for 10 to 15 percent of all childhood injuries. These include fractures, dislocations and soft tissue injuries. The most common sites for fractures are the distal forearm and hand, accounting for 50 percent of pediatric fractures. The peak incidence is during adolescence.

Management Goals

1. Suspicion of fracture—get relevant history of trauma. Try to judge velocity of injury: High/low
2. Examine the child without further jeopardizing the fracture
3. Splint the limb
4. Order relevant X-rays in two orthogonal planes
5. Provide analgesia and sedation to calm the child and thus the agitated family
6. Send appropriate referral
7. Suspect (Red flag) for child abuse, pathological fractures, e.g. osteogenesis imperfecta, when multiple fractures seen, or fractures through bone tumors when fractures occur spontaneously or after trivial injury (Fig. 15.6.1), or in metabolic derangements like rickets, scurvy, etc.

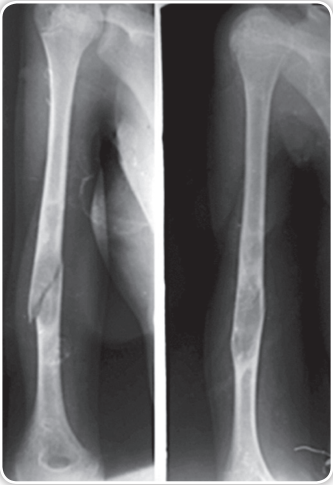

Figure 15.6.1 Pathological fracture through a simple bone cyst in the humeral diaphysis

Injuries in the Appendicular Skeleton: Common Upper Extremity Injuries

Fall on the Outstretched Hand

The usual mechanism is a fall on the outstretched hand with the hand or volar aspect of the wrist hitting the ground first. In such cases suspect the following:

1. Distal radius and/or distal ulna fracture (Figs 15.6.2 and 15.6.3)
2. Diaphysis of radius and ulna fracture

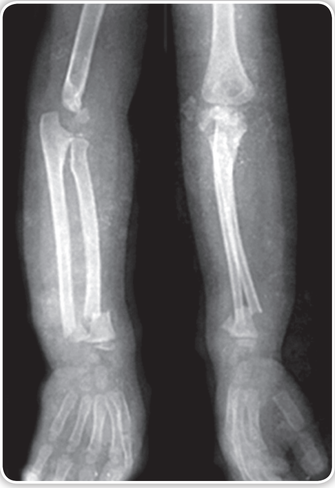

Figure 15.6.2 Displaced distal end radius and ulna fracture

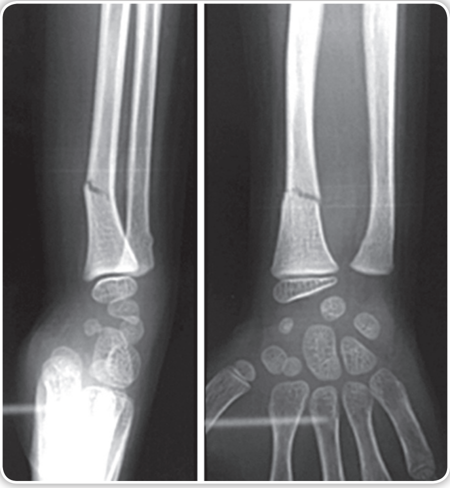

Figure 15.6.3 Angulated greenstick fracture of the distal radius with a buckle (torus) fracture of the distal ulna

3. Fracture with dislocation:
   - i. *Monteggia type*: Fracture of proximal ulna with dislocation of the radial head (Fig. 15.6.4)
   - ii. *Galleazi type*: Fracture of distal radius with dislocation of distal radio-ulnar joint

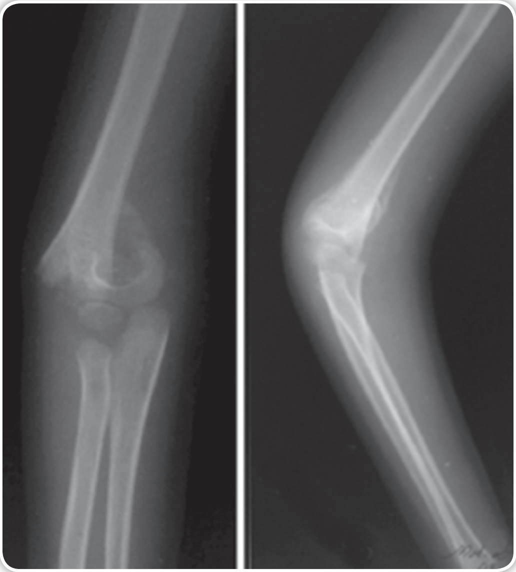

Figure 15.6.4 Monteggia fracture dislocation. Note the ulnar fracture associated with dislocated radial head. The associated dislocation necessitates early manipulative reduction, hence early referral is indicated

4. Fractures around the elbow:
    i. Supracondylar humerus fractures (Fig. 15.6.5)
    ii. Lateral condyle humerus fractures (Fig. 15.6.6)
5. Proximal humerus fracture (Fig. 15.6.7)
6. Fracture clavicle (Fig. 15.6.8).

Fall Directly on the Elbow

If the injury is with a fall directly on the elbow, then suspect the following:

1. Supracondylar humerus fractures.
2. *Lateral condyle of distal humerus fractures*: This is a fracture of necessity, i.e. it requires urgent attention if displaced due to its intra-articular and physeal extension of the fracture pattern.

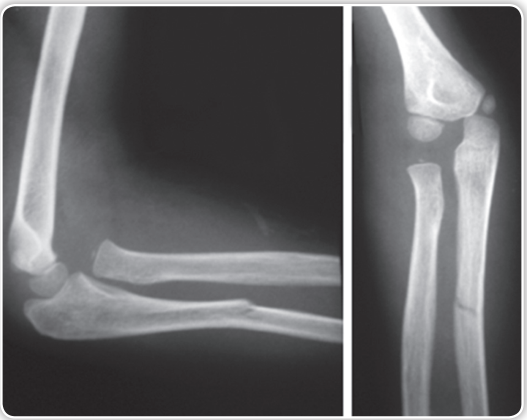

Figure 15.6.5 Displaced supracondylar fracture of distal humerus

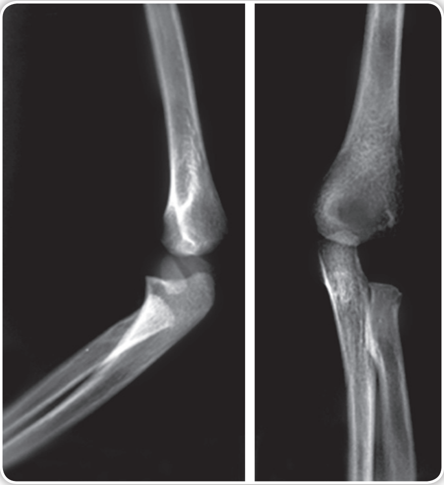

Figure 15.6.6 Displaced lateral condyle humerus fracture

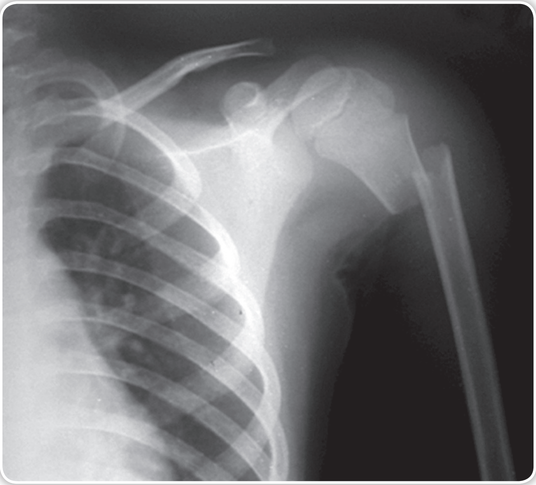

Figure 15.6.7 Displaced proximal humerus fracture

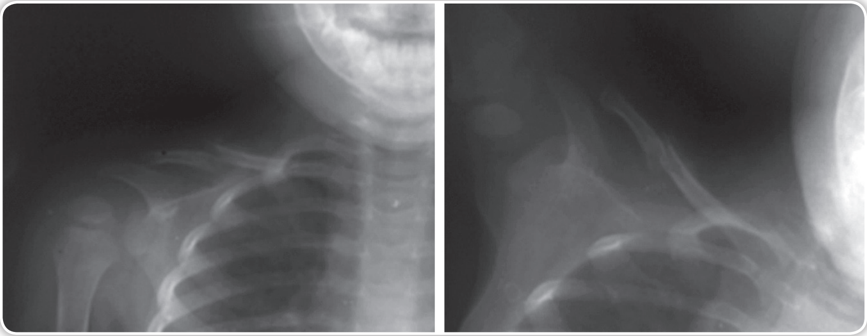

Figure 15.6.8 Clavicle fracture

If the injury is due to a sudden pulling of the arm/hand of the child, then suspect the following.

"Pulled elbow" synonym nursemaid's elbow: It is essentially a partial tear of the annular ligament around the neck of the radius, resulting in subluxation of the proximal radioulnar joint.

Check for radial pulse and sensorimotor deficit especially for median and ulnar nerve dysfunction. Urgent referral is necessary as delay can cause vascular compromise. Avoid tight bandaging as that may lead to compartment syndrome and later Volkmann's ischemic contracture in the forearm.

Lateral condyle fractures are epiphyseal injuries and displaced ones require urgent operative reduction, so as to obtain union and good elbow function.

Immobilization Methods and Optimum Position for Injuries to the Upper Limb

1. A simple cuff and collar sling or a triangular swathe around the neck to support the involved side elbow in flexion can be used for clavicular fractures, fractures of the humeral shaft, the distal humerus.
2. An Above elbow posterior back slab with the elbow in flexion and forearm in the mid prone position is most suitable for fractures around the distal humerus, for fractures of the shaft of the radius and ulna, and for the fracture dislocations in the forearm.
3. A Below elbow posterior back slab is suitable for fractures around the distal radius, and in hand fractures.

Injuries in the Appendicular Skeleton: Common Lower Extremity Injuries

The tibia is the most commonly fractured bone, followed by the femur. Fractures around the hip and pelvis account for less than 1 percent of pediatric fractures and hence have not been included in this chapter. Figures 15.6.9 to 15.6.12 are showing various fractures of the lower limb.

Femur fractures entail a significant amount of blood loss, hence the child should be admitted for observation, ruling out other injuries and hemodynamic stabilization. Splintage with a Thomas splint or surface skin traction helps relive the muscle spasm associated with the injury.

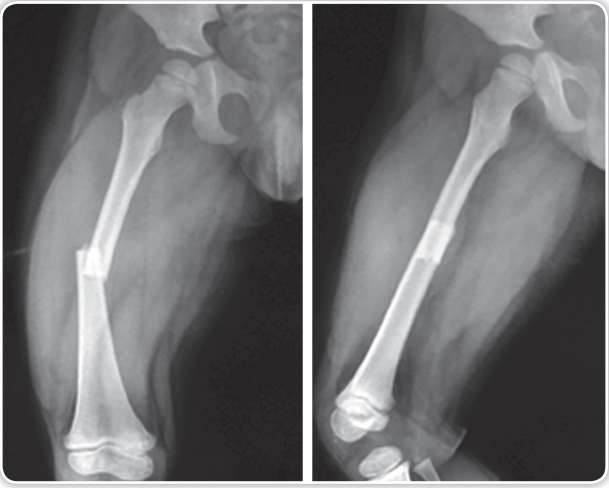

Figure 15.6.9 Transverse fracture of the femoral diaphysis in a 4-year-old child, with bone overlap

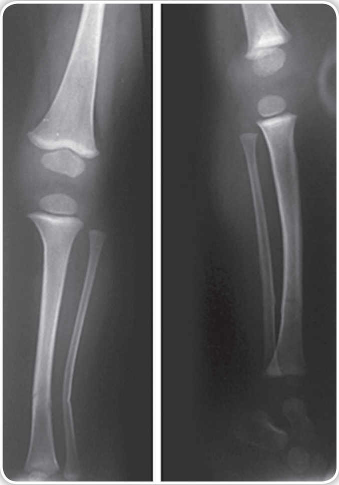

Figure 15.6.10 Greenstick fracture of tibia and fibula with valgus angulation

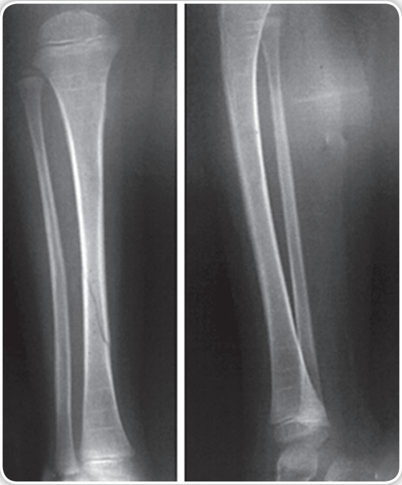

Figure 15.6.11 Undisplaced spiral fracture of the tibia, the fracture line is not visualized on the lateral projection but is well visualized on the anteroposterior projection. This highlights the need for X-rays in two orthogonal planes

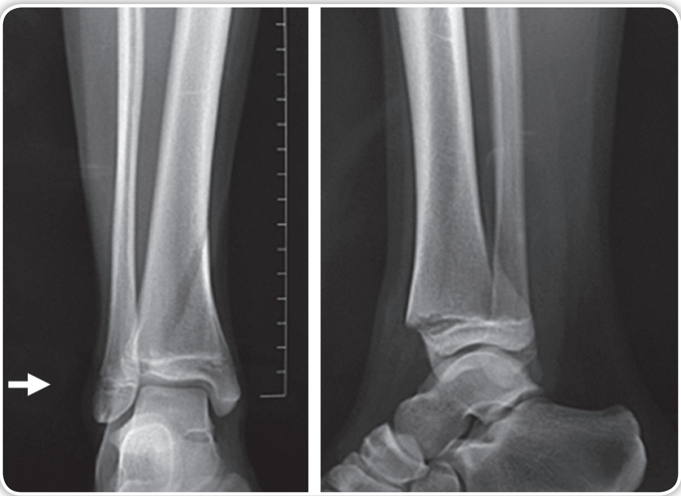

Figure 15.6.12 An epiphyseal fracture separation of the lower end of tibia in an adolescent child

Immobilization Methods and Optimum Position for Injuries to the Lower Limb

1. An above knee posterior back slab with the knee in 15° of flexion and ankle in 90° position is suitable for fractures of the diaphysis of the tibia and fibula, as well as distal femur.
2. A Thomas splint or surface skin traction is required for immobilization of fractures of the femur shaft, as well as proximal femur.
3. A below knee posterior back slab is suitable for fractures around the distal tibia, ankle injuries and in foot fractures.

Injuries to the Axial Skeleton

Injuries to the spine extending from the cervical to the lumbosacral region and fractures of the rib cage are included in this section. Such fractures need to be suspected in children who have sustained high velocity trauma, e.g. polytrauma due to road accidents, falls from height, etc. In such situations head injury and associated visceral injuries and their management take priority.

Definitive fracture management is usually deferred and the axial skeleton often immobilized with collars, belts till the child is treated for the head injury and/or visceral injuries.

Injuries to the children need to be treated properly at the earliest opportunity so as to ensure optimum results. A high index of suspicion should be maintained to come to a conclusive diagnosis and appropriate treatment or referral instituted.

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16

Otolaryngological Emergencies

Jaydeep Choudhury

16.1 OTOLARYNGOLOGICAL ISSUES

Jaydeep Choudhury

Pain in the ear is a very common complaints in children. This often causes incessant cry and disturbed sleep. The common causes of ear pain or otalgia are listed in Table 16.1.1.

TABLE 16.1.1
Common causes of ear pain in children

Source of pain	*Causes*
Intrinsic to external ear	Otitis externa
	Foreign body
	Trauma
	Perichondritis infected preauricular cyst or sinus
Intrinsic middle ear and mastoid	Acute otitis media
	Middle ear effusion
	Barotrauma
	Mastoiditis
Referred	**Via trigeminal nerve:**
	1. Dental
	2. Temporomandibular joint
	3. Jaw
	4. Oral cavity

Contd...

Contd...

	Via facial nerve:
	1. Herpes zoster
	2. Bell's palsy
	Via glossopharyngeal nerve:
	1. Tonsil
	2. Oropharynx
	3. Nasopharynx
	Via vagus nerve:
	1. Laryngopharynx
	2. Esophagus
	3. Thyroid
	Via cervical nerves:
	1. Lymphnodes
	2. Cervical spine
	3. Neuralgia
	Others:
	1. Migraine
	2. Sinuses
	3. Salivary gland
	4. CNS

Acute Otitis Externa

Acute otitis externa is an inflammatory condition, anatomically which involves external auditory canal and external surface of the tympanic membrane. Pathologically there is a compromise of the lining of the canal.

Management

1. Cleaning and debridement of the ear canal with cotton swab and hydrogen peroxide.
2. An acidifying agent like 2 percent acetic acid is useful to inhibit growth of bacteria and virus.
3. Topical preparations—mixed antibiotics, anti-fungal and steroid drops or antibiotics alone atleast for 3 days after resolution of symptoms. Steroids decrease inflammation.
4. For pain relief—nonsteroidal anti-inflammatory drugs (NSAIDs).

Prevention

1. Drying the ear canal after bathing and swimming
2. Use of ear plugs while swimming
3. Instillation of 2 percent acetic acid solution in the ear.

Acute Otitis Media (AOM)

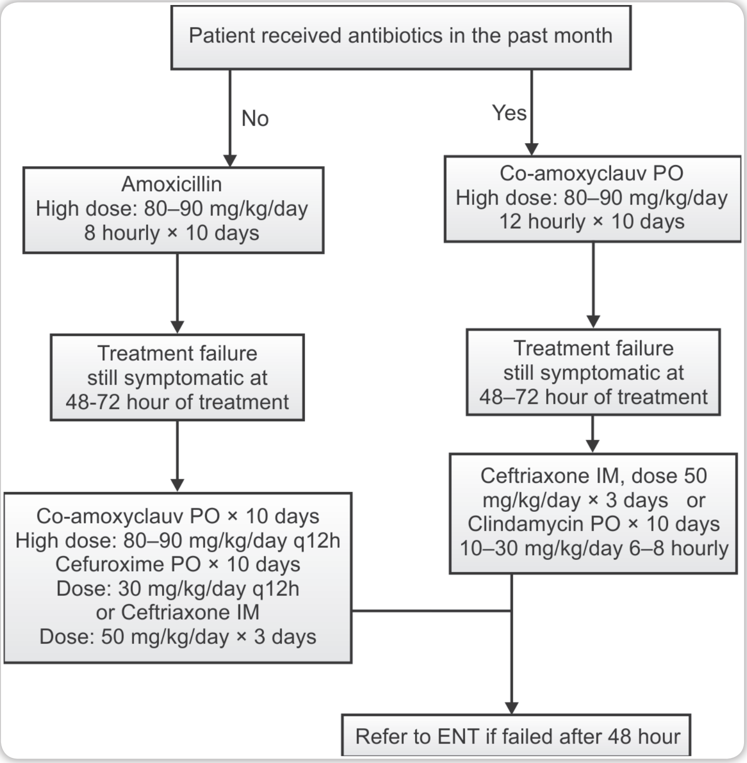
Acute otitis media is characterized by acute onset of pain in the ear, fever and otorrhea with middle ear effusion.

Management

Treatment algorithm of AOM is shown in Flow chart 16.1.1.

In children with penicillin allergy, **e**rythromycin 50 mg/kg/day 6 hourly or cotrimoxazole 6 to 12 mg, trimethoprim 30 to 60 mg, sulfamethoxazole 40 mg/kg/day 12 hourly.

Flow chart 16.1.1 Treatment of acute otitis media



Foreign Body in the Nose and Ear

Children love to play with small objects and put those in their nose and ears. The foreign bodies found in nose and ears are parts of toys, various food particles, pieces of papers, beads and sometimes small insects.

Management

The following are the prerequisites for foreign body removal in an emergency department:

1. The child should be restrained properly or adequately sedated, otherwise the attempts will cause unnecessary trauma
2. A good light source is very important for proper visualization
3. The following instruments should be available—nasal speculum, alligator forceps, curette and suction apparatus

The following techniques may be adopted for foreign body removal:

1. Manually grasping the object with forceps
2. Getting behind the object with a curette, when there is room for advancing the curette
3. Irrigation by water
4. Applying suction to the surface of the foreign body.

One should attempt foreign body removal in the emergency only if one is confident for removal because repeated failed attempts result in increased swelling and trauma. It may also push the object to such a location from where it will be very difficult to remove. An attempt for removal is based on the characteristic of the foreign body and anatomic location.

1. A live insect: It should first be killed with either 2 percent lidocaine or mineral oil.
2. Vegetable matter or sponges: Irrigation should be avoided because the added water will make the foreign body swell.
3. Smooth round: Suction should be applied.
4. Large occlusive nasal foreign body: Bag and mask ventilation over mouth and manually occluding the noninvolved nostril.

If attempted removals are unsuccessful, one should refer to an otolaryngologist.

Epistaxis

The rich blood supply and thin mucus membrane makes the nose vulnerable to bleed spontaneously following minor trauma. Most of the time bleeding from nose or epistaxis is mild and self-limited.

Management

Minor Bleeding

1. Pinching the nostrils together for 5 to 10 minutes nonstop. One should not release the grip in between.
2. A piece of gauze soaked with nasal decongestant epinephrine 1:10,000 or phenylephrine induces local vasoconstriction.

Major Bleeding

1. When anterior nasal bleeding is difficult to control: Cautery or anterior nasal packing with petroleum jelly may be done.
2. Posterior nasal bleeding: It is less amenable to cautery. It requires urgent admission for posterior nasal packing or epistaxis balloon. These interventions should be done by an otolaryngologist.

Sinusitis

Sinusitis is a common form of upper respiratory tract infection in children. Children usually do not present with classical features of sinusitis like the adults. It is very difficult to distinguish between simple viral sinusitis, rhinosinusitis and acute bacterial sinusitis.

Acute Bacterial Sinusitis

Among the various causes of sinusitis in children acute bacterial sinusitis requires urgent treatment. Upper respiratory tract infections with symptoms persisting for more than 10 days without much improvement are more likely to be bacterial sinusitis. Viral sinusitis presents with fever, constitutional symptoms and mucoid nasal discharge. But bacterial sinusitis classically presents with high fever and purulent nasal discharge. Children often have cough which is worse at night or on lying down.

Treatment

Antibiotics are the mainstay of treatment. Initially broad spectrum antibiotics like high dose amoxicillin or amoxicillin-clavulanic acid should be started. If penicillin resistance is suspected azithromycin or cefuroxime may be started. The duration of treatment should be 10 to 14 days.

Oral Cavity Lesions

Lesions in the oral cavity can be very distressing in children. It causes severe discomfort and pain. Children refuse to eat and may cry incessantly.

Herpetic Gingivostomatitis

It is caused by Herpes simplex virus (HSV). Commonly seen in children 6 months to 5 years.

Treatment

1. Topical: Anesthetics like diphenhydramine syrup mixed 1:1 solution with magnesium hydroxide. This should be applied to the affected area every 2 hours. Topical acyclovir has no role.
2. Paracetamol and ibuprofen for fever and pain relief.
3. Acyclovir: Should be started within 72 hours of the onset of lesions.
4. Due to severe pain children often refuse oral intake and they are at risk of dehydration. Oral intake of fluid should be encouraged or else intravenous fluid may be given.

Herpangina

It is an oropharyngeal lesion caused by coxsackieviruses A and B. Commonly seen in children 3 to 10 years. It is highly contagious. Skin infected with coxsackieviruses A manifests as hand, foot and mouth (HFM) disease.

Systemically there are flu-like symptoms of fever, sore throat and headache. The painful vesicles and ulcerations occur over the posterior pharynx and tonsils.

The disease is self-limited, resolving within 3 to 7 days. Treatment is symptomatic with systemic antipyretics and topical analgesics.

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17

Dermatological Emergencies

Mahasweta Chaudhuri, Jayanta Bandyopadhyay, Amiya Kumar Mukhopadhyay

17.1 FEVER WITH RASH

Mahasweta Chaudhuri, Jayanta Bandyopadhyay

The common causes of fever with rashes in children are summarized in Table 17.1.1.

TABLE 17.1.1
Conditions associated with fever with rashes

Cause	*Conditions*
Viral	Varicella, measles, rubella, roseola, erythema infectiosum, HFM (Hand, foot and mouth disease—usually due to coxsackie B virus), enteroviral exanthem, HSV infection, infectious mononucleosis, viral hemorrhagic fever (like dengue), hepatitis B
Bacterial	Scarlet fever, scalded skin syndrome, toxic shock syndrome, meningococcemia, infective endocarditis, spirochetal infections (syphilis), leptospirosis, lyme disease
Inflammatory diseases	Systemic lupus erythematosus, systemic onset juvenile rheumatoid arthritis, vasculitis syndrome (Kawasaki disease, Henoch- Schönlein purpura), inflammatory bowel disease
Malignancy	Leukemia, lymphoma
Miscellaneous	Rickettsial, fungal, helminthic and protozoal infections. Stevens-Johnson syndrome, toxic epidermal necrolysis, histiocytosis syndrome

Different Forms of Skin Rash

i. Macule—flat and impalpable
ii. Papule—raised and circumscribed, palpable
iii. Vesicle—raised, circumscribed, filled with fluid, less than 0.5 cm diameter
iv. Pustule—raised lesions filled with pus
v. Petechiae—flat or raised hemorrhagic, up to 0.5 cm diameter, non-blanching
vi. Purpura—flat or raised hemorrhagic spots over 0.5 cm in diameter
vii. Ecchymosis—large, irregular discolored areas, resulting from extravasated blood.

Figures 17.1.1 to 17.1.3 show children with measles, chicken pox and meningococcal rash respectively. The conditions presenting with various characteristic rashes is shown in Table 17.1.2.

Distribution

i. Generalized—Measles, scarlet fever, disseminated HSV
ii. Central—Varicella, roseola, Still's disease
iii. Peripheral—Gionatti Crosti syndrome, HMF, late stage of Kawasaki
iv. Flexural areas—Scarlet fever, SSS
v. Extensor surface—HSP, erythema nodosum, late stage of erythema infectiosum
vi. Localized—Lyme disease, herpes zoster, anthrax, streptococcal erysipelas, pseudomonal ecthyma gangrenosum.

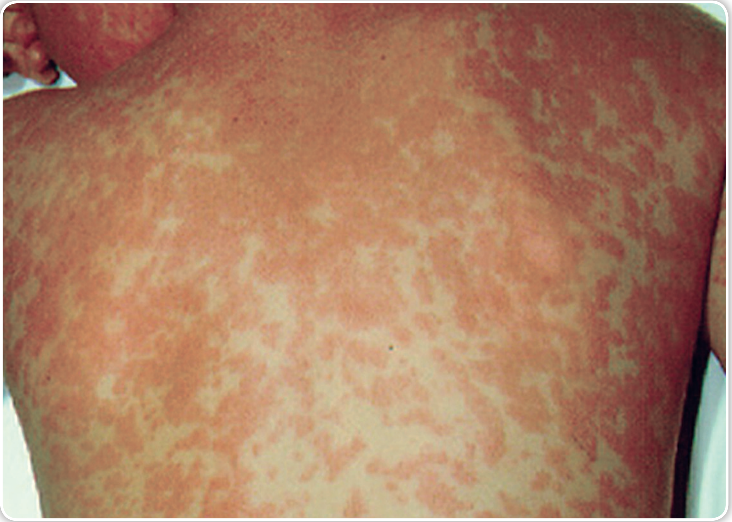

Figure 17.1.1 A child with measles

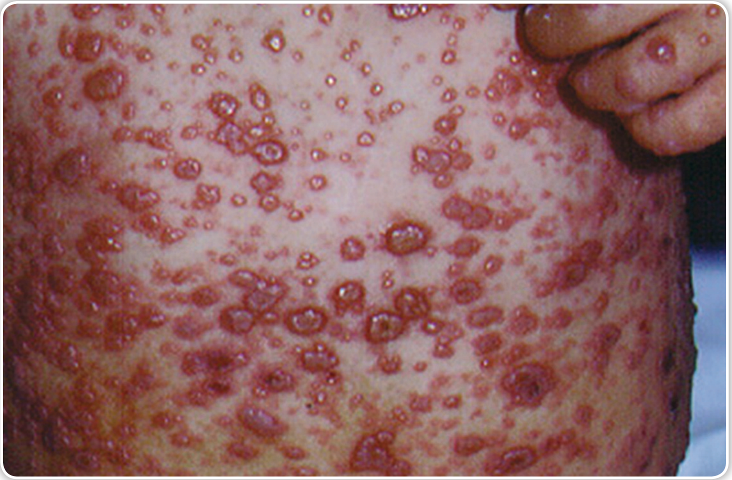

Figure 17.1.2 A child with chickenpox

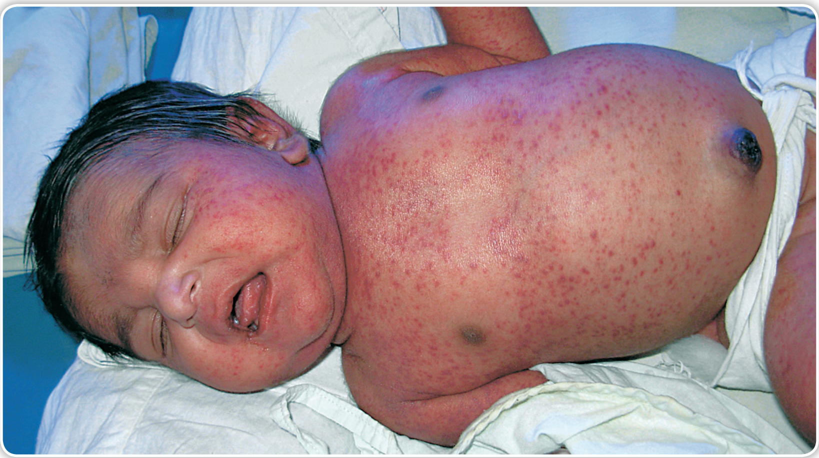

Figure 17.1.3 A child with meningococcal rash

Management

Most viral exanthematous fevers are self-limiting and hence symptomatic and supportive measures are enough.

Unwell Child with Fever and Petechiae/Purpura

At least 90 percent of children with fever and petechiae will not have meningococcal disease. TSS has been dealt separately in other chapter. However, recognition and early treatment of the child with meningococcal

TABLE 17.1.2

Differential diagnosis of rashes

Erythematous	*Maculopapular*	*Petechiae or Purpura*	*Vesico-bullous*
Staphylococcal SSS Toxic shock syndrome Anaphylaxis TEN Scarlet fever	Viral exanthem (measles, roseola, rubella) Urticaria Lyme disease Pityriasis Drug reaction Erythema multiforme Stevens-Johnson syndrome Meningococcemia Rocky Mountain spotted fever Eczema, Psoriasis	Meningococcemia ALL HSP TTP ITP Vasculitis	Varicella Zoster HSV DIC Pemphigus vulgaris Bullous pemphigus Necrotizing fascitis Contact dermatitis HFM disease Stevens-Johnson syndrome

disease is paramount. Clinical signs and laboratory investigations will help determine those who should be treated for suspected meningococcal disease.

Indicators of meningococcal disease (or other serious bacterial infection) include:

i. Unwell child with abnormal vital signs and poor peripheral perfusion
ii. Altered conscious state
iii. Purpura more than 2 mm, nonblanching, maculopapular rash becomes apparent within first 24 hours of disease; sparsely distributed on face, trunk and lower extremities. Later the petechiae in center of macules become hemorrhagic
iv. Abnormal blood indices (including WCC $>15 \times 10^9$/L and $<5 \times 10^9$/L and raised CRP >8 mg/dL). Though blood culture and CSF analysis including culture should be sent but one should not wait for reports. It is ideal to send baseline serum cortisol level.

Principles in the Management of Meningococcal Disease

i. Immediate fluid resuscitation, early administration of antibiotics (third generation cephalosporin—cefotaxime 200 mg/kg/day or ceftriaxone 100 mg/kg/day or penicillin 2.5 to 4 lakh U/kg/day) and involvement of intensive care staff
ii. Any child with meningococcal disease, including one who appears nontoxic, may deteriorate rapidly
iii. Endotracheal intubation to be considered early
iv. Give methylprednisolone 30 mg/kg before (or within 30 minutes) first dose of antibiotics

v. Consider hydrocortisone 1mg/kg 6 hourly if cortisol level is low, otherwise may cause Addisonian crisis

vi. Contact chemoprophylaxis: It is important that prophylaxis be given within 24 hours to all intimate, household or day care contacts who have been exposed to index case within 10 days of onset:

   a. Infants and children >1 month of age—rifampicin 10 mg/kg orally 12 hourly (max 600 mg) for 2 days
   b. Adults—rifampicin 600 mg 12 hourly for 2 days
   c. Infants less than 1 month of age—rifampicin 5 mg/kg 12 hourly for 2 days
   d. Pregnancy/contraindication to rifampicin—ceftriaxone 125 mg (less than 12 years)/250 mg (more than 12 years) intramuscularly as a single dose.

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17.2 URTICARIA AND ANGIOEDEMA

Amiya Kumar Mukhopadhyay, Jayanta Bandyopadhyay

Urticaria manifests as transient smooth slightly elevated erythematous and edematous patches (wheals) often associated with severe pruritus. The typical features are shown in Figures 17.2.1 and 17.2.2. Angioedema manifests as localized swelling of skin and mucosa with characteristic stinging sensation.

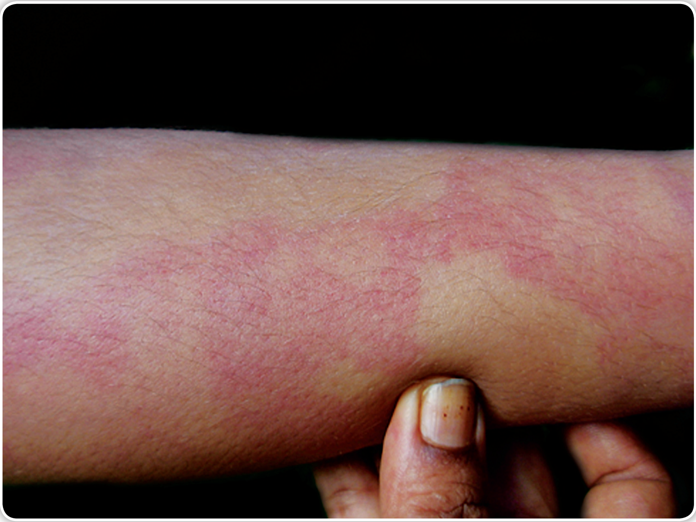

Figure 17.2.1 Urticaria

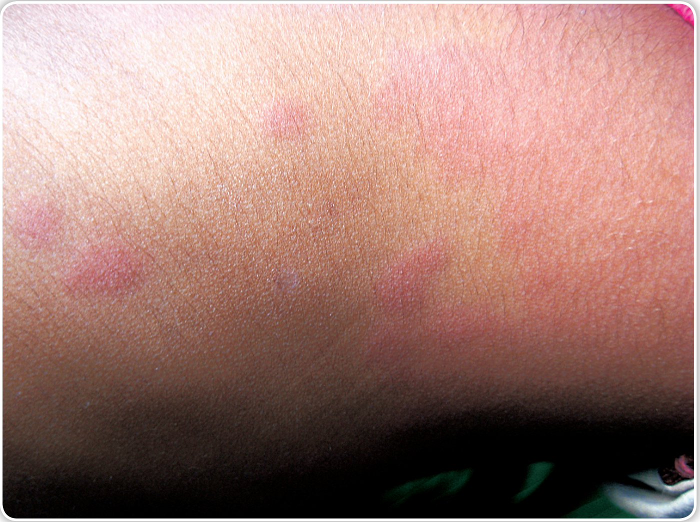

Figure 17.2.2 Typical wheal in urticaria

Management

1. Remove the responsible agent. Drugs like aspirin, NSAID, sulfonamides, penicillins, etc. Foods like nuts, chocolates, seafood, etc
2. Hospitalize children with acute urticaria and angioedema as they may suffer from respiratory obstruction. One should be ready for tracheostomy if severe laryngeal edema develops
3. If severe utricaria develops with low blood pressure, tachycardia and features of shock, management should be like anaphylaxis
4. Counseling and reassurance is an important aspect of management.

Moderate to Mild Urticaria

1. Oral antihistaminics are the mainstay; sometimes injectable antihistaminics may be required. Antihistaminics used in children are highlighted in Table 17.2.1. In acute urticaria, 5 to 10 days course is needed
2. H2 blocker
3. Oral or parenteral steroid. Methylprednisolone, prednisolone or dexamethasone may be used
4. Topical soothing agents like calamine lotion.

Chronic Urticaria

1. Antihistaminics, may be given for 3 to 6 months
2. H2 blocker
3. Cyproheptadine—especially in cold urticaria
4. Montelukast
5. Doxepine hydrochloride
6. Calcium channel blockers
7. Desensitization of allergens.

TABLE 17.2.1

Antihistaminics used in children for urticaria and angioedema

Drug	*Dose*
Sedative	
1. Promethazine	0.5 mg/kg/dose every 8 hourly
2. Chlorpheniramine	0.35 mg/kg/day
3. Hydroxyzine	1–2 mg/kg/day
4. Cyproheptadine	0.2 mg/kg/day
Mild sedative Cetrizine	0.25 mg/kg/day
Nonsedative	
1. Fexofenadine	30 mg 12 hourly (6–12 years)
2. Loratadine	5 mg/day (used above 2 years)

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17.3 STEVENS-JOHNSON SYNDROME (SJS)

Amiya Kumar Mukhopadhyay

Stevens-Johnson Syndrome (SJS) compromises extensive erythema multiforme (EM) of the trunk and mucous membrane accompanied by fever, myalgia and arthralgia. The lesions are typically known as target lesions as shown in Figures 17.3.1 and 17.3.2.

Management

1. All the children should be hospitalized
2. If any drug is the precipitating factor, it should be stopped. This is the most important step
3. Basic investigations—complete blood count, electrolytes, liver and renal function tests

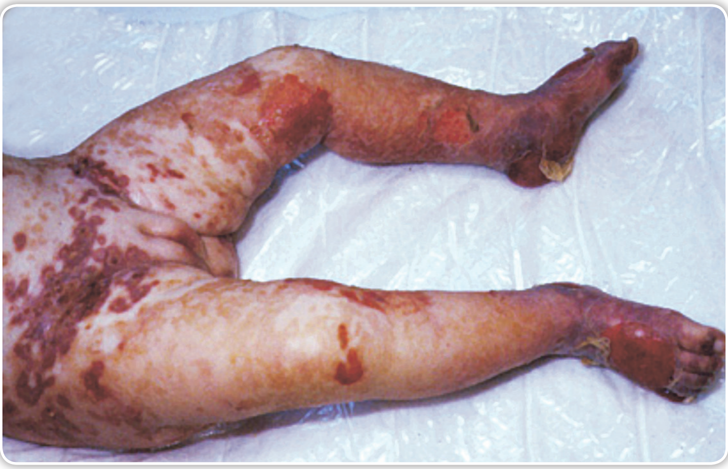

Figure 17.3.1 Erythema multiforme

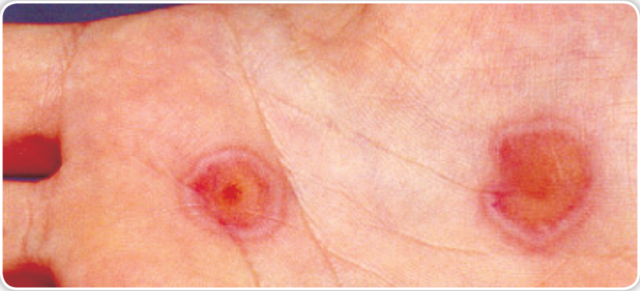

Figure 17.3.2 Target lesion

4. Thermoneutral environment should be maintained. Ideal temperature is environmental temperature at 30 to 32°C
5. Due to extensive skin and mucus membrane involvement, these children may not be able to take adequate fluid and are prone to dehydration. They should be given intravenous fluids. Minimum urine output in children should be 1 mL/kg/h
6. Role of corticosteroid is controversial. Injection methylprednisolone or dexamethasone may be given
7. Antihistaminics and analgesics may give some symptomatic relief
8. Healing process may take about 2 weeks, so proper skin care is very important. Topical emollients and antibiotics may give some relief, but topical silver sulfadiazine should be avoided because of its causative association. Use topical agents such as 0.5 percent silver nitrate solution or 0.05 percent chlorhexidine solution to cleanse the skin. Warm these solutions before application
9. H2 blockers like ranitidine should be given to prevent stress ulcer.

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17.4 TOXIC EPIDERMAL NECROLYSIS

Amiya Kumar Mukhopadhyay

Toxic epidermal necrolysis (TEN) is a severe life-threatening disorder with rapidly progressive eruption of the skin usually following drug intake which leads to generalized loss of epidermis and mucosa, resembling burn. The typical features are shown in Figures 17.4.1 and 17.4.2.

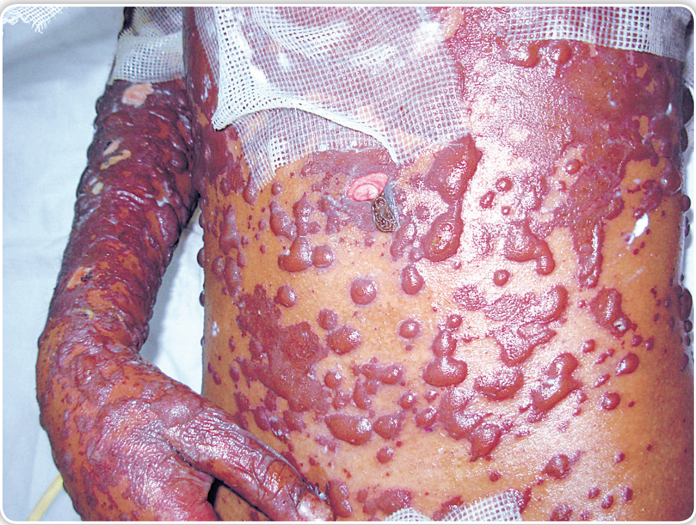

Figure 17.4.1 Typical features of TEN

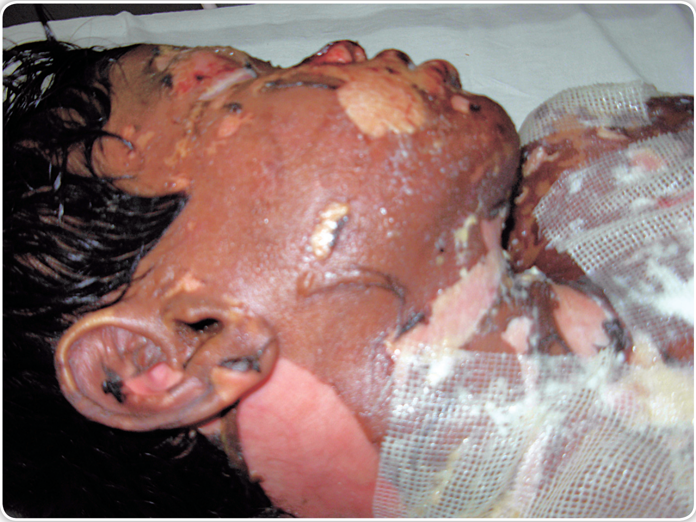

Figure 17.4.2 TEN affecting face

Management

1. Early hospitalization, ideally in a burn unit
2. The suspected drug should be stopped and structurally related compound is to be avoided
3. Adequate IV fluid and maintenance of fluid and electrolyte balance
4. Oral prednisolone, though controversial, may be given at 2 to 4 mg/kg/day for 3 to 4 days
5. IV Immunoglobulin or cyclosporin A may be given
6. Prophylactic antibiotic may be given if large area is involved. But one should be careful about selection of antibiotic as many antibiotics may precipitate
7. Skin and ophthalmologic care should be taken. Skin is a protective barrier. Large scale affection of skin may cause improper thermoregulation. Topical antibiotics may also cause allergic reaction and produce fever. One should be very cautious in selection of topical agents. The crucial factor is maintenance of strict asepsis without much intervention.

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18

Infections

Jayanta Bandyopadhyay, Jaydeep Choudhury

18.1 FEVER WITHOUT FOCUS

Jayanta Bandyopadhyay, Jaydeep Choudhury

Infants and children are often brought to the emergency department with fever as the only complain.

Stabilization

Any febrile child should be stabilized first and then clinically evaluated before a decision is taken to send them home.

i. Child should be made comfortable by removing clothing and excess blankets and clothing
ii. Antipyretic like paracetamol 10 to 15 mg/kg may be given orally
iii. Oral fluid intake should be encouraged
iv. Children who have high temperature and those who run the risk of having febrile convulsion may be given tepid water sponging to bring down the temperature.

Approach

Diagnostic approach to a child 0 to 3 months age with fever without any focus is shown in Flow chart 18.1.1. The observation items to identify a child with SBI are shown in Table 18.1.1. Approach to a child with fever without focus 3 to 36 months is shown in Flow chart 18.1.2.

Flow chart 18.1.1 Approach to fever with no focus in 0 to 3 months

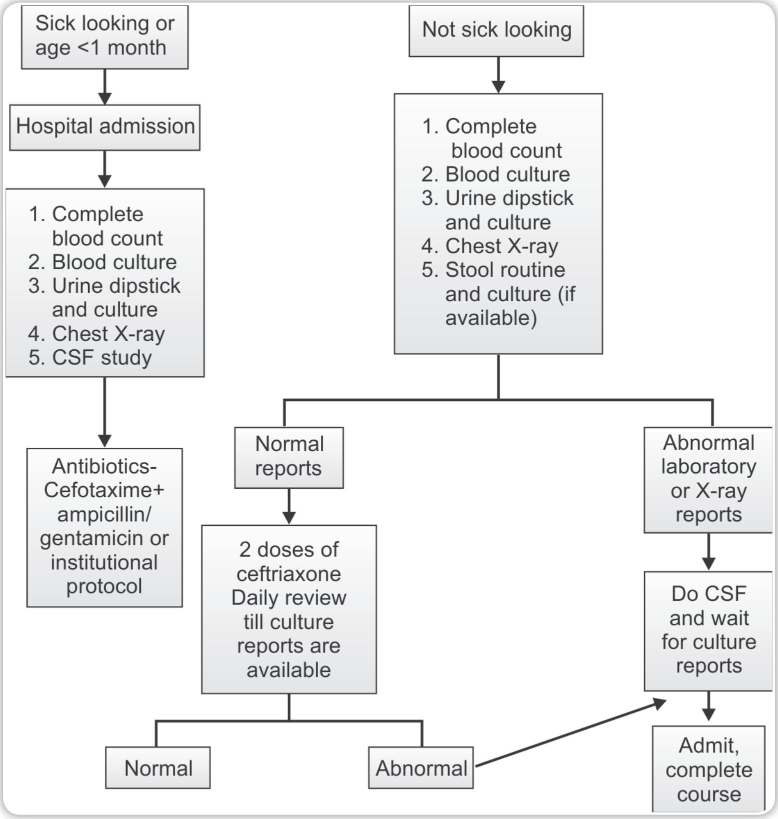
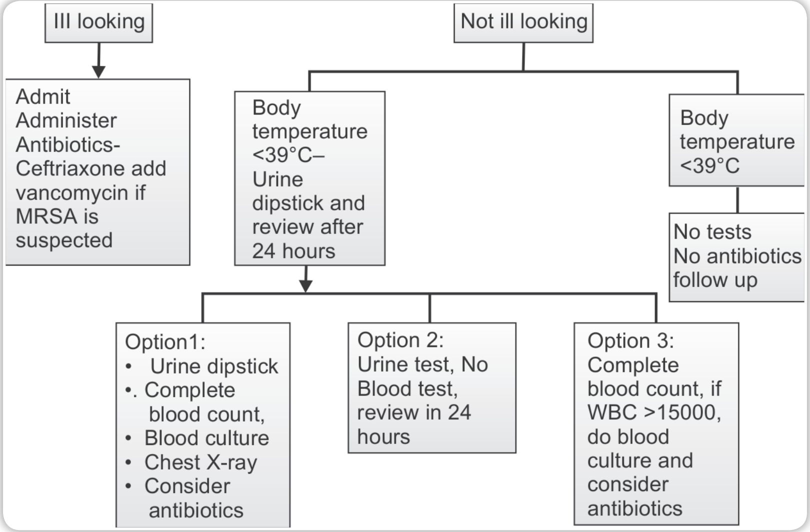

Sick looking or age <1 month
Hospital admission
1. Complete blood count
2. Blood culture
3. Urine dipstick and culture
4. Chest X-ray
5. CSF study
Antibiotics- Cefotaxime+ ampicillin/ gentamicin or institutional protocol
Not sick looking
1. Complete blood count
2. Blood culture
3. Urine dipstick and culture
4. Chest X-ray
5. Stool routine and culture (if available)
Normal reports
2 doses of ceftriaxone Daily review till culture reports are available
Normal
Abnormal
Abnormal laboratory or X-ray reports
Do CSF and wait for culture reports
Admit, complete course

TABLE 18.1.1
Observation items to identify a child with SBI

Item	*Unwell*	*Very unwell*
Appearance	Sick looking (lethargy, reduced activity)	Absent eye contact, doesn't recognize parents, no activity
Quality of cry	Whimpering	Weak cry, high pitched cry
Response to cuddling	Slow response, unwilling	Too weak to respond
Alertness	Drowsy	Frequently falls sleep, difficult to wake
Hydration	Slightly dry mouth	Dry mouth, sunken fontanelle, doughy skin
Color	Peripheral cyanosis or pallor	Mottled pale face or ashen
Sociability/ stimulation	Briefly smiles and responds	Not smiling, anxious face, expressionless

Flow chart 18.1.2 Approach to a child with fever without focus 3 to 36 months



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18.2 SEVERE AND COMPLICATED MALARIA

Jaydeep Choudhury

Severe malaria is almost always due to *Plasmodium falciparum*, though recently some *Plasmodium vivax* cases are also presenting with complications. Complicated malaria is a medical emergency with high mortality. However appropriate timely management can limit morbidity and mortality. Irrespective of etiology all complicated malaria with severe manifestations should be treated with injectable antimalarials.

Management

Supportive

1. Maintenance of ABC. Proper positioning with attention to airways
2. Oxygen should be given and respiratory support if necessary
3. Care of eyes, mucosa and skin
4. IV fluid. In case of shock, fluid resuscitation with normal saline or Ringer's lactate should be done. Under or over hydration should be avoided. Blood sugar should be monitored and appropriate dextrose containing fluid should be given and corrections made if required
5. Nasogastric tube should be placed *in situ* to minimize the risk of aspiration
6. Convulsion should be treated appropriately
7. Hyperpyrexia should be managed accordingly
8. Monitoring of vital signs: Preferably 4 hours till the patient is out of danger. Intake output chart should also be maintained
9. Antibiotic coverage should be given as these children prone to septicemia.

Antimalarial Chemotherapy

1. Child should be weighed and dose of antimalarials should be calculated according to body weight
2. Initially antimalarials should be given as injections and it should be replaced by oral preparations as soon as the child is able to take oral medicines
3. If intravenous preparations are not available, crushed tablets may be given through nasogastric tube, but it may cause vomiting and inadequate drug absorption
4. Antimalarial chemotherapy—artesunate or artemether.

Artesunate: 2.4 mg/kg IV stat dose and then at 12, and 24 hours, followed by once daily for 7 days. Artesunate, 60 mg per ampoule is dissolved in 0.6 mL 5 percent sodium bicarbonate diluted to 3 to 5 mL with 5 percent dextrose and given immediately after reconstitution by IV bolus. If the child is able to swallow, the daily dose may be given orally. As soon as the child is able to swallow tetracycline 4 mg/kg/dose 4 times daily in children above 8 years or doxycline 3.5 mg/kg once daily in children above 8 years should be given for 7 days. In children below 8 years, clindamycin 20 mg/kg/day in 2 divided doses should be given for 7 days.

Artemisinin based combination therapy (ACT): Those who can swallow, a fixed dose of artemether lumefantine can be given orally for 3 days as shown in Table 18.2.1.

Poor Prognostic Indicators

i. High parasite density—more than 5 percent RBC infected or parasite density >250000/μL
ii. If mature parasites are more—more than 20 percent parasites contain visible pigments
iii. More than 5 percent peripheral leukocytes contain visible malaria pigment.

Management of Complications

There may be several complications of falciparum malaria. Mortality is high unless these complications are managed early.

Severe Anemia

Anemia develops due to acute destruction of red blood cells, this is more common with hyperparasitemia. The following conditions warrant packed cell transfusion:

TABLE 18.2.1
Arteminsinin based combination therapy (ACT)

Item	*Unwell*	*Very unwell*
Appearance	Sick looking (lethargy, reduced activity)	Absent eye contact, doesn't recognize parents, no activity
Quality of cry	Whimpering	Weak cry, high pitched cry
Response to cuddling	Slow response, unwilling	Too weak to respond
Alertness	Drowsy	Frequently falls sleep, difficult to wake
Hydration	Slightly dry mouth	Dry mouth, sunken fontanelle, doughy skin
Color	Peripheral cyanosis or pallor	Mottled pale face or ashen
Sociability/ stimulation	Briefly smiles and responds	Not smiling, anxious face, expressionless

i. PCV less than 12 percent or hemoglobin below 4 g/dL
ii. Transfusion is indicated in less severe anemia associated with respiratory distress, impaired consciousness and high parasite load (>20% RBC infected).

Hypoglycemia

The following situations may precipitate hypoglycemia:

i. Children below 3 years with hyperparasitemia or having seizures
ii. Children treated with quinine.

Blood sugar should be monitored frequent as the features of hypoglycemia may be difficult to distinguish clinically.

Cerebral Malaria

It may evolve gradually or occasionally, it may progress very rapidly to convulsion or coma. Convulsion may be subtle also. Good supportive care is essential along with management of coma. Children may also present with features of raised intracranial tension. These children should be managed with general measures like head end elevation, fluid restriction and hyperventilation by mechanical ventilation. Mannitol is not indicated.

Hyperpyrexia

It is common in children and may lead to convulsion. Tepid sponging, paracetamol and cool ambient temperature are the mainstay of treatment.

Hyperparasitemia

This is typically seen in nonimmune children with severe disease. Exchange transfusion or cytapheresis may be considered as parasite load is high.

Lactic Acidosis

Clinically deep breathing with chest retractions without much auscultatory findings are characteristic. It usually accompanied cerebral malaria, anemia and dehydration. Acid-base status should maintain and hypovolemia, anemia, seizure should be controlled.

Circulatory Collapse

Gram-negative septicemia should be excluded in case of circulatory collapse and should be treated accordingly with fluids and antibiotics.

DIC

Should be treated with vitamin K and blood.

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18.3 DENGUE

Jaydeep Choudhury

The following are the four clinically recognized syndromes:

1. Undifferentiated fever
2. Dengue fever (DF)
3. Dengue hemorrhagic fever (DHF)
4. Dengue shock syndrome (DSS), it is actually a severe form of DHF.

The following are the tests:

1. The ELISA tests (IgM capture ELISA and IgG capture ELISA)
2. Rapid immunochromatographic card tests.

The interpretation of dengue IgM and IgG in various dengue infections are shown in Table 18.3.1.

Management

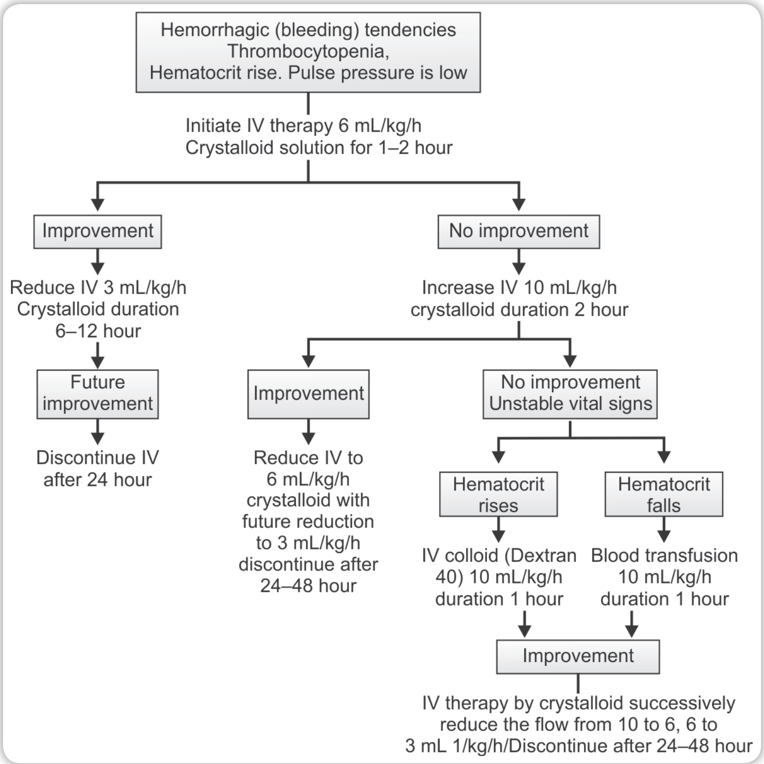
Volume replacement of dengue fever and dengue hemorrhagic fever in minimal set up is shown in Flow chart 18.3.1. For further information, refer to the chapter on shock.

TABLE 18.3.1

Interpretation of dengue IgM and IgG

IgM	*IgG*	*Interpretation*
Negative	Negative	Early sample
Negative	Positive (low titer)	Postdengue infection
Negative	Positive (high titer)	Secondary dengue infection
Positive	Negative	Primary dengue infection
Positive	Positive (low titer)	Current or recent dengue infection
Positive	Positive (high titer)	Secondary dengue infection

Flow chart 18.3.1 Volume replacement flow chart of dengue fever and dengue hemorrhagic fever


Hemorrhagic (bleeding) tendencies
Thrombocytopenia,
Hematocrit rise. Pulse pressure is low
Initiate IV therapy 6 mL/kg/h
Crystalloid solution for 1–2 hour
Improvement
No improvement
Reduce IV 3 mL/kg/h
Crystalloid duration
6–12 hour
Increase IV 10 mL/kg/h
crystalloid duration 2 hour
Future improvement
Improvement
No improvement
Unstable vital signs
Discontinue IV after 24 hour
Reduce IV to 6 mL/kg/h crystalloid with future reduction to 3 mL/kg/h discontinue after 24–48 hour
Hematocrit rises
Hematocrit falls
IV colloid (Dextran 40) 10 mL/kg/h duration 1 hour
Blood transfusion 10 mL/kg/h duration 1 hour
Improvement
IV therapy by crystalloid successively reduce the flow from 10 to 6, 6 to 3 mL 1/kg/h/Discontinue after 24–48 hour

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18.4 COMMUNITY ACQUIRED PNEUMONIA

Jaydeep Choudhury

Inflammation of lung parenchyma which occurs in previously healthy child and the infection is acquired outside hospital environment is community acquired pneumonia (CAP). The typical chest X-rays of pneumonia are shown in Figures 18.4.1 to 18.4.3.

Management

1. Check O_2 saturation. Aim is to keep more than 92 percent saturation.
2. Antibiotics: The choice of antibiotic is mainly empirical depending on the age, severity of illness, etiology, local epidemiology and possible drug resistance pattern. The choice of antibiotics for outpatient and injectable medications are shown in Tables 18.4.1 and 18.4.2, respectively.

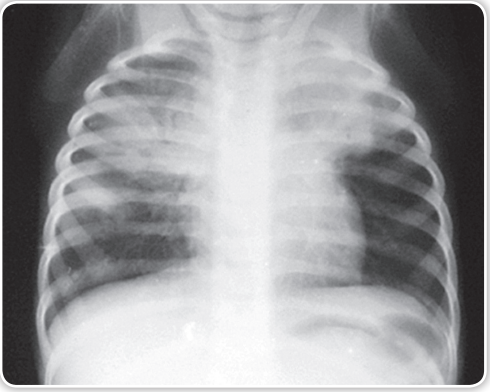


Figure 18.4.1 Bilateral upper lobe consolidation

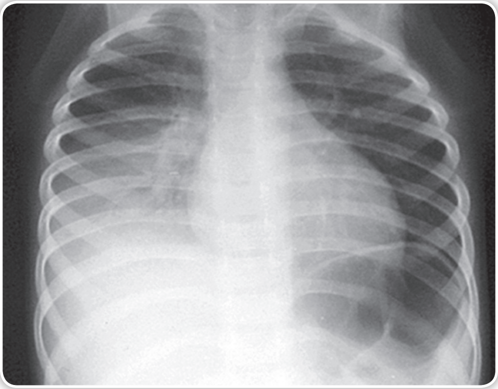
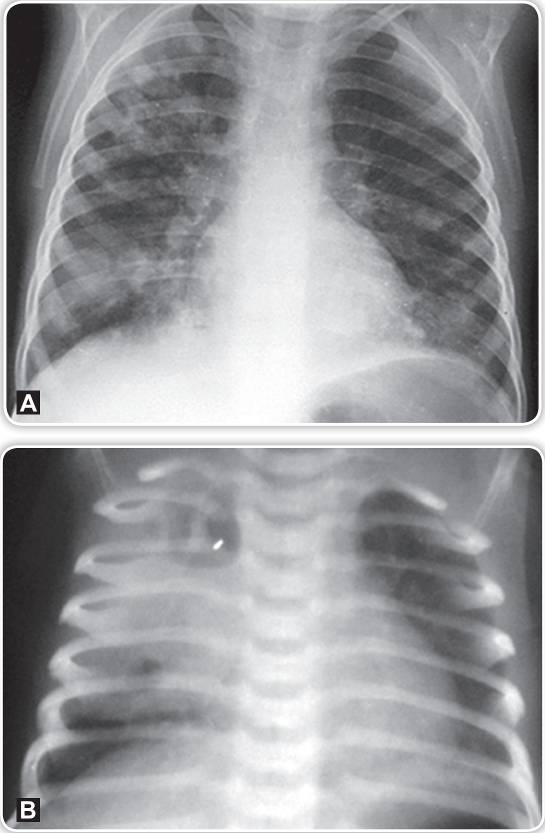


Figure 18.4.2 Right basal lobe consolidation



Figures 18.4.3A and B Bronchopneumonia

TABLE 18.4.1

Antibiotics for outpatient treatment of CAP

Age	*First line*	*Second line*
3–5 months	Amoxicillin	Co-amoxyclav
>5 months	Amoxicillin	Macrolides / co-amoxyclav

TABLE 18.4.2

Injectable antibiotics for inpatient treatment of CAP

Age	*First line*	*Second line*
<3 months	Cefotaxime/ceftriaxone	Add aminoglycoside
3 months to 5 years	Co-amoxyclav/ampicillin	Co-amoxyclav/cefotaxim/ ceftriaxone
>5 years	Ampicillin/co-amoxyclav/ macrolide	Cefotaxime/ceftriaxone/ macrolide
Suspected staphylococcus	Cefuroxime/co-amoxyclav/3rd generation cephalosporin	Ceftriaxone/cefotaxime/ vancomycin/linezolid

The duration of treatment should be 7 to 10 days. *Staphylococcus pneumonia* without complications should be treated for 2 weeks and those with complications for 4 to 6 weeks.

Neonatal pneumonia and any pneumonia below the age of 3 months should always be admitted and treated with injectable antibiotics.

The dose of various antibiotics is as follows:

1. Ampicillin 100 to 200 mg/kg/day every 6 hourly
2. Amoxicillin 30 to 50 mg/kg/day every 8 hourly, high dose 60 to 80 mg/kg/day
3. Cefotaxime 150 mg/kg/day every 8 hourly
4. Ceftriaxone 100 mg/kg/day every 12 hourly
5. Erythromycin 40 mg/kg/day every 6 hourly
6. Clarithromycin orally 15 mg/kg/day every 12 hourly
7. Azithromycin orally 10 mg/kg/day once daily for 5 days.

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18.5 ACUTE MENINGITIS

Jayanta Bandyopadhyay, Jaydeep Choudhury

Pyogenic meningitis in children has to be suspected early, diagnosed early and treated promptly.

Management

1. Maintenance of ABC
2. Restore circulating volume and urinary output
3. Monitor vital signs, hydration and neurological status
4. If impaired consciousness keep patient on parenteral fluid and nutrition only
5. Start antibiotics after collecting CSF report as soon as possible.

Antibiotic Therapy

Full Term Neonates <1 Week

Ampicillin 150 mg/kg/day IV every 8 hourly with cefotaxime 150 to 200 mg/kg/day IV every 8 to 12 hourly.

1 Week to 3 Months

Ampicillin 200 mg/kg/day IV every 6 hourly with cefotaxime 150–200 mg/kg/day IV every 6 hourly.

Children >3 Months

Cefotaxime 200 mg/kg/day IV every 6 hourly.

Add Vancomycin

60 mg/kg/day IV every 6 hourly in the following situations:

1. Gram stain showing gram positive cocci
2. Very ill child with hemodynamic instability.

Once the organism and sensitivities are recognized switch to appropriate and narrowest spectrum antibiotic.

Duration of Antibiotics

Neonates—2 weeks (3 if gram-negative organism)
S. pneumoniae—10 to 14 days

H. influenzae 7—10 days
N. meningitides 5—7 days
Gram-negative infections—3 weeks.

Supportive Care and Monitoring

1. Input and output chart and daily weight
2. Daily head circumference
3. Watch for SIADH—urine specific gravity and serum sodium
4. IV fluids—maintenance (If SIADH restrict to 2/3 maintenance)
5. Anticonvulsant if convulsions.

Prophylaxis

1. *S. pneumoniae*—Not indicated
2. *H. infuenzae*—Refer to chapter on acute epiglottitis
3. *N. meningitidis*—Refer to chapter fever with rash
4. Contact with patient's oral secretions (within 7 days of presentation)
5. Health care workers: Only if mouth resuscitation, endotracheal intubation or has been in direct contact with the patient's oral secretions. No prophylaxis for indirect contact.

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18.6 URINARY TRACT INFECTION

Jaydeep Choudhury

Urinary tract infection (UTI) is diagnosed when urine culture report reveals a colony count more than 10^5 colony forming unit (CFU) per mL of clean catch urine. This signifies significant bacteriuria.

Treatment

Indication for Admission

1. Neonates and infants
2. Children of any age with high fever and/or flank pain, sepsis or shock
3. Known complex underlying urological pathology
4. Persistent vomiting, dehydration or inability to take oral medication
5. Known/suspected causative organism resistant to oral medication
6. Psychosocial issues: inability of family to care for child appropriately.

Antibiotic Therapy

Bacterial antibiotic resistance patterns are geographically determined and should be reviewed at each hospital to determine the best initial oral antibiotics. Using broader spectrum antibiotics might contribute in emergence of resistant organisms.

Agents that are excreted in the urine but do not achieve therapeutic concentrations in the bloodstream, such as nalidixic acid or nitrofurantoin, should not be used to treat UTI in febrile infants and young children in whom renal involvement is likely. The recommended antibiotics for treatment of UTI in children are shown in Tables 18.6.1 and 18.6.2.

Intravenous antibiotics can be switched to oral antibiotics once the causative agent and the antibiotic sensitivities were identified. The patient can be discharged home in the following circumstances:

1. More than 2 months of age
2. Afebrile for >24 hours
3. Tolerating oral fluids.

Duration of Therapy

About 7 to 10 days treatment regimens are recommended for UTI, longer duration up to 14 days might be necessary in complicated UTI.

The follow-up investigations are shown in Table 18.6.3.

MCUG can be performed once infection is cleared. DMSA should be done 4 to 6 weeks after an UTI episode.

TABLE 18.6.1

Antimicrobial for oral treatment of UTI

Antimicrobial	*Dosage*
Trimethoprim-sulfamethoxazole	4–6 mg TMP, 20–30 mg SMX/kg/day q12 hours
Amoxicillin/co-amoxyclav	20–40 mg of amoxicillin/kg/day in 3 doses
Cephalexin	50–100 mg/kg/day in 4 doses
Cefixime	8 mg/kg/day in 2 doses
Cefuroxime axetil	10 mg/kg/day in 2 doses
Cefpodoxime	10 mg/kg/day in 2 doses

TABLE 18.6.2

Antibiotics for parenteral treatment of UTI

Antibiotic	*Daily Dosage*
Ceftriaxone	75 mg/ kg/day every 12 hours
Cefotaxime	150 mg/kg/day divided every 6 hours
Ceftazidime	150 mg / kg/ day divided every 6 hours
Gentamicin	7.5 mg/kg/day every 12–24 hours
Amikacin	15 mg/kg/day divided every 12 hours
Ampicillin	100 mg / kg/ day divided by every 6 hours

TABLE 18.6.3

Follow-up investigations of UTI

Investigation	Indication	Rationale
Ultrasound abdomen	All children following UTI	To rule out major urinary tract structural pathology
MCUG	1. Acute pyelonephritis	To rule out vesicoureteric reflux
	2. First UTI in a boy or girl <2 years age	Posterior urethral valve in boys
	3. First UTI in a child of any age with family history of UTI, urinary tract abnormalities or abnormal voiding pattern	Anatomical or functional bladder abnormalities
DMSA Scan	Suspected pyelonephritis at young age Recurrent UTI evidence of vesicoureteric reflux	Rule out renal scarring
Radionuclide cystogram	Used to follow up vesicoureteric reflux after initial traditional MCUG	Advantage: Less radiation Disadvantage: Not as sensitive as MCUG does not show urethral Or bladder abnormalities

TABLE 18.6.4

Prophylactic antibiotics for prevention of UTI

Prophylactic antibiotics	*Dosage*
Trimethoprim-sulfamethoxazole	2 mg TMP once daily
Nitrofurantoin	1 mg/ kg dose once daily
Cephalexin	25 mg/kg/dose once daily

Prophylactic Antibiotics

Although the evidence of benefit of long-term low-dose antibiotic prophylaxis for prevention of UTI is not strong, it is the most widely used strategy to prevent UTI in clinical practice. Antibiotic prophylaxis is recommended in the following:

i. Infants with UTI pending completion of evaluation
ii. Children with VUR and
iii. Those with recurrent febrile UTI even if the urinary tract is normal.

Medications used for prophylaxis are usually given as single bedtime dose (Table 18.6.4).

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19

Toxic Shock Syndrome

Mahasweta Chaudhuri, Jayanta Bandyopadhyay

19.1 TOXIC SHOCK SYNDROME

Mahasweta Chaudhuri, Jayanta Bandyopadhyay

Toxic shock syndrome (TSS) is a multisystem disease manifested by sudden onset of fever, chills, hypotension, and rash with multisystem involvement. Differences between Staphylococcal and Streptococcal TSS are shown in Table 19.1.1. The diagnostic criteria of Staphylococcal and Streptococcal TSS are shown in Table 19.1.2.

Management

Seriously ill patients may require care in the ICU, including dialysis for renal failure, ventilatory support for acute respiratory distress syndrome (ARDS), and correction of coagulopathy using plasma and blood products as necessary. Treatment regimens for toxic shock syndrome (TSS) includes the following:

Staphylococcal Toxic Shock Syndrome

1. Aggressive fluid support with normal saline or colloids
2. Vasopressor/inotrope infusion as necessary
3. Antistaphylococcal antibiotics (penicillin and clindamycin or vancomycin and clindamycin in patients with penicillin allergy)
4. Removal of tampons, nasal packing, and other foreign objects
5. Intravenous immunoglobulin (IVIG) therapy
6. Aggressive supportive care in an ICU.

TABLE 19.1.1

Differences between staphylococcal and streptococcal TSS

Findings	*Staphylococcal TSS*	*Streptococcal TSS*
Age	15–35 years	20–50 years
Sex	More common in females	Males and females
Causes	1. Menstruation. 2. Nonmenstrual conditions: Surgical wounds, such as hernia repair and arthroscopy, respiratory infections, varicella infection, nasal packing.	1. Skin infections 2. Surgical wounds 3. Pharyngitis 4. Varicella infection 5. Influenza virus infection 6. NSAIDs:
Local invasive disease	Absent	Present
Generalized erythroderma	Present	Absent
Nausea, vomiting, or diarrhea	>90% of patients	Uncommon
Bacteremia	Uncommon	60% of patients
Toxins implicated	TSST-1; enterotoxins B and C	Streptococcal pyrogenic exotoxins A and B
Mortality rate	3.3%	30%

TABLE 19.1.2

Diagnostic criteria of staphylococcal and streptococcal TSS

Staphylococcal TSS	*Streptococcal TSS*
Major criteria (all required)	**Hypotension plus two or more**
1. Temperature >38.8°C	1. Impairment
2. Hypotension (orthostatic/shock)	2. Coagulopathy
3. Rash (erythroderma with late desquamation)	3. Hepatic involvement
Minor criteria (any three)	4. Adult respiratory distress syndrome
1. Mucous membrane inflammation	5. Generalized erythematous macular rash
2. Vomiting, diarrhea	6. Soft tissue necrosis
3. Liver abnormalities	**Definite case**
4. Renal abnormalities	Clinical criteria plus group A streptococcus from a normally sterile site (blood, CSF, pleural/peritoneal fluid, tissue biopsy specimen)
5. Muscle abnormalities	
6. Central nervous system abnormalities	
7. Thrombocytopenia	**Probable case** Clinical criteria plus group A streptococcus from a nonsterile site (throat, vagina, sputum or superficial skin lesions)
Exclusionary criteria	
1. Absence of another explanation	
2. Negative for blood cultures (except for *S. aureus*)	

Streptococcal Toxic Shock Syndrome

1. Normal saline or colloids may be used. Intractable hypotension that results from diffuse capillary leaking may require large amounts of these fluids. Albumin replacement may be necessary in patients in whom albumin levels drop lower than 2 g/dL
2. Vasopressor/inotrope infusion as necessary
3. Surgical debridement is mandatory in the presence of tissue necrosis
4. Use penicillin plus clindamycin for antibiotic treatment. Clindamycin therapy for streptococcal toxic shock syndrome produces better results than penicillin alone
5. Studies have suggested a salutary effect of IVIG therapy
6. Aggressive supportive care in an ICU is needed
7. The role of hyperbaric oxygen in streptococcal toxic shock syndrome remains uncertain in the absence of any comparative clinical trials.

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20

Medicolegal Issues

Jaydeep Choudhury

20.1 MEDICOLEGAL ISSUES IN EMERGENCY ROOM

Jaydeep Choudhury

Emergency room services actually deals with patients whose life is at risk. Thus, it is potentially a volatile area, particularly when the patient is a child. Here the children are brought with some acute symptoms and the caregivers expect prompt action and relief. On the other hand, sometimes, it is not possible to pinpoint a diagnosis. In such a situation the attending doctor is expected to be gentle, caring and confident. A good emergency duty doctor not only treats the disease well but also counsels the parents avoiding any misunderstanding or communication gap. It is imperative that doctors are answerable for the outcome of the patients and are bound to justify the actions taken by them. Most of the time more than medical mismanagement, it is the lack of communication and improper dealing of the situation that leads to litigation. In such a scenario the medicolegal aspect of emergency services should always be protected.

Documentation in Emergency Ward

Proper documentation with date, time, name and signature is evidence which justifies that the patient was attended to at the emergency, treatment given and diagnosis was attempted. Documentation is of utmost importance in clinical practice.

Consent in Emergencies

In emergency situations consent may not be taken for providing life-saving treatment, even though the nature of treatment may amount to adventure. Even emergency operations like tracheostomy may be performed without consent if it saves the life of the patient. But the doctor should do only that, which is necessary to save the life, ensures improvement and prevent deterioration. Nonurgent problems should not be dealt with in the emergency.

Whenever possible consent should be obtained from the parents or relative. Indian Penal Code states that a consent given by a child under 12 years of age is invalid. Indian Majority Act states that a person who has attained the age of 18 years may give legally valid consent for medical or surgical procedures.

Medical Negligence

It is failure on part of a doctor to exercise skill and diligence which are required of a professional. It may be either an act of omission or an act of commission which an emergency doctor of average skill is not supposed to do.

Criminal negligence is the act of negligence on part of the practitioner who is so careless or reckless that it almost results in severe damage to the health of the individual.

In emergency situations patients are brought in critically ill, often moribund condition. In such a situation the priority is to save the life without bothering about complications. A typical example, is rib fracture following cardio-respiratory resuscitation. Here the relatives cannot sue the treating doctor for rib fracture.

Informing the Police

Police should be intimated in the following situations:

i. Unnatural death and sudden death after treatment or reaction to medicine
ii. Unknown, unconscious patient
iii. Any poisoning
iv. Motor vehicular and other accidents that account for fractures, head injuries, accidental falls needing inpatient treatment
v. Burns
vi. Drowning
vii. Tetanus, gas gangrene, significant violence needing inpatient treatment
viii. Attempted suicide, poisoning and attempted homicide. In attempted poisoning the doctor should collect the stomach wash and blood samples clotted and in EDTA and hand it over to police
ix. Human, animal or snake bite
x. Abandoned child
xi. Suspected child abuse
xii. Rape, minor’s pregnancy, MTP, battered baby.

Breaking Bad News

If a junior doctor is on duty it is better to call a senior colleague for breaking a bad news. The parents should be taken to a separate room or place and make them sit before breaking the news. The communication should be made as reassuring as possible. A second opinion is always welcome in such a situation. Often the parents become very emotional and angry. Both the extremes of situation should be dealt calmly and with empathy.

Death Certificate

Proper judgement for issue or denial of death certificate is often very crucial.

Child Brought Dead who was Under Treatment at the Hospital

1. If the child was under treatment at the hospital for some chronic and potentially fatal disease and has been brought dead to the emergency, death certificate has to be issued if the death seems to be a logical outcome of the disease
2. Death certificate can be issued if no foul play is suspected
3. But if the cause of death seems unnatural then the police has to be informed.

Child Brought Dead by the Parents

After initial assessment and attempts for resuscitation death has to be declared. Police has to be informed and postmortem advised.

Child Brought Dead by a Mob

1. Rapid assessment whether the child is really dead.
2. It is prudent to start an attempt for resuscitation as this will not only help the physician to buy time but also pacify an anxious crowd.
3. The signs of death should be reassessed infront of a few members of the crowd and death declared. Police has to be informed and postmortem advised.
4. If the mob seems to be rowdy, the police should be informed and asked for protection before declaring death.

Caution has to be exercised, if a child is suddenly brought dead or succumbs in the emergency department shortly after initiating treatment. Doctor should not yield to pressure or comply on sympathetic ground. It is prudent to plead helplessness to the relatives and inform the police. Here again proper communication and tact is of utmost importance. If the situation is such that it is not possible to tackle the situation alone, then help of colleagues should be taken.

In an emergency department, treatment cannot be refused to any child. Not only the attending physician, but the whole paramedical team should be motivated to get involved. Confusion arises when two or more critically ill children are brought to the emergency at the same time. One should be careful to assess and attend to all of them. It is important to quickly examine and assess who is the most critical of them and treat him or her, still not denying the basic management to the rest of the sick children. Here the paramedical team plays a crucial role. The accompanying family members should not feel that basic treatment is being denied to their child.

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